ARCHIVES OF OPHTHALMOLOGY.

A CASE OF TRAUMATIC, AT FIRST DOUBTFUL, ORBITAL SARCOMA, FOLLOWED BY ASEPTIC THROMBOSIS OF THE CAVERNOUS SINUS.

SUCCESSFUL SINUS OPERATION BY DR. FRANK HARTLEY. DEATH LATER BY THE EXTENSION OF THE PSEUDOPLASM. AUTOPSY. REMARKS.[1]

By Dr. HERMAN KNAPP, New York.

(*With five figures, the first four in text-plates VI.–IX., the fifth in the text.*[2])

Francis H. Cuffe, æt. thirty, New York City, admitted to New York Ophthalmic and Aural Institute, January 1, 1900.

History. — Five weeks previously the end of a billiard cue struck the ball and upper orbital margin of the left eye. The eye watered considerably for about ten days, without any other symptoms. The lids and conjunctiva began to swell and the eyeball protruded. Soon after, it receded somewhat, but since December 25, 1899, the protrusion has been more pronounced.

Condition on Admission.—The lids are swollen; the eye is pushed forward and down; its mobility restricted, most so upward, least outward; patient counts fingers at 20′; F. T. and pupil normal; media clear; periphery of optic disk and adjacent retina swollen; veins engorged, dark red (thrombosis of central vein?); no hemorrhage; arteries and veins pulsate on strong pressure; the upper lid hangs as an inert, dark-red mass of flesh over the eyeball; it feels soft on the nasal side, hard and somewhat uneven on the temporal; the hardness begins at the brow and extends uninterruptedly about 15 *mm* down; the patient is

[1] The first part of this article was communicated to the American Ophthalmological Society, May 2, 1900.

[2] Fig. 1 is from a photograph by Dr. Ch. M. Stevens, of New York, the three others from photographs by Dr. J. B. Solley, New York.

slightly apathetic, but gives reasonable answers when questioned. For years he has been hard of hearing on both sides; his drum-heads are pale, irregular, cicatricial, free from inflammation, but giving evidence of former purulent otitis media; the right ear is completely deaf; the left moderately hard of hearing.

The **probable diagnosis** is: injury of the roof of the orbit, possibly a fissure, with effusion of blood and serum; yet as there is no subconjunctival hemorrhage noticeable, the presence of a traumatic sarcoma is thought of. The patient is ordered to lie in bed, with sustaining diet.

Course of Disease.—*January 2d.*—Considerable chemosis; temporal margin of od free; the other parts ill-defined and swollen; on palpation no tenderness on any part of the orbit.

January 5th.—Fingers at 5′; less swelling of lids and conjunctiva.

January 6th.—Fingers at 8′; veins of retina dark and swollen; arteries and veins pulsate on light, and empty on strong, pressure.

January 8th.—Od and retina more swollen; chemosis greater on lower side; fingers at 4′, excentrically.

January 9th.—Neuro-retinitis; movements of hands only. Incision into nasal part of the upper lid liberates little serum.

January 10th.—Pupil large; fundus reflex normal; no details recognizable; left eyeball immovable.

January 19th.—Hearing of left ear weaker; the swelling of the lids and conjunctiva slowly increasing.

January 21st.—The skin of the left side of the face, the left anterior half of the forehead and scalp, and the anterior surface of the left auricle show diminution of sensibility; cornea sensitive; dull in lower part, which is exposed.

January 30th.—Keratitis from lagophthalmos.

February 18th (see Fig. 1).—The condition of the lid and conjunctiva essentially the same; patient more apathetic; the whole region of skin supplied by the first division of the fifth pair now anæsthetic; the cornea is sloughing; iris prolapsed in lower part; temp. 98.6°; pulse 68, normal in character; sensibility of mucous membrane of mouth and nose preserved. From right ear slight discharge at times; from left none. Fundus, S., and F. of right eye normal; no signs of inflammation in either ear.

Extended Clinical Diagnosis.—Œdema of the lids and conjunctiva, exophthalmos; diminution and gradual aboli-

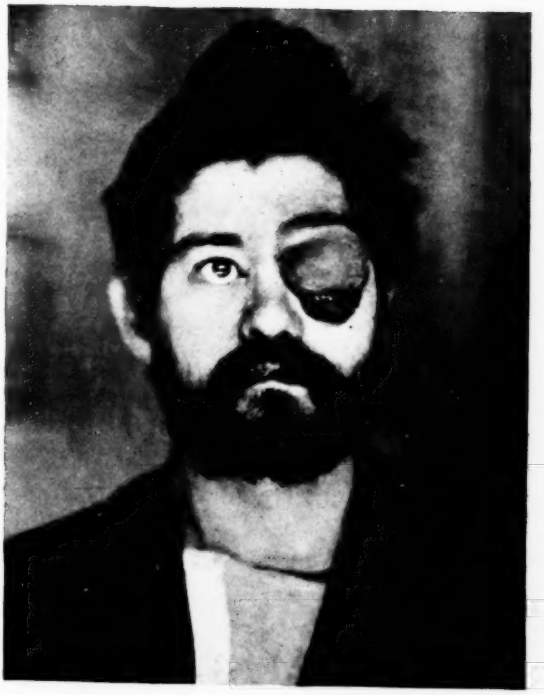

FIG. 1.

tion of the mobility of the third, fourth, sixth, and anæsthesia of the first division of the fifth pair; the venous engorgement and œdema of the retina and the neuro-retinitis furnish sufficient evidence to assume the presence of thrombosis of the left cavernous sinus. As the most probable cause I assumed either a hematoma, compressing the ophthalmic vein, or a fissure of the roof of the orbit with injury to the sinus in a similar way as an arterio-venous communication in the cavernous sinus is brought about by a traumatic rupture of the carotid in the sinus, yet I thought that the presence of that rare and fatal disease of a traumatic palpebral and orbital sarcoma could not be excluded. In the latter case all surgical interference would be contra-indicated; in the former, the chances, according to our present knowledge, were scarcely less appalling. In the limited number of cases reported, the other eye nearly always became blind under the same symptoms as the first, namely, by extension of the thrombus from the cavernous sinus of one side to the other. Panas,[1] who collects the incident literature, counter-advises an operation, as only one case had been operated on before (by Horsley) and the patient had died eleven hours after the operation. Yet in our case I did not want to let misfortune continue its inexorable course, but was determined to have an operation performed as soon as there were symptoms of beginning thrombosis in the other eye. These symptoms soon enough made their appearance.

February 21st.—I made the round through the hospital at 2 P.M., as usual, examined our patient, particularly his right eye, for which I now cared chiefly. I found everything normal. At 4:30 P.M. the same day I showed him to a visiting physician. He presented a different picture in the right eye: œdema of conjunctiva, lids, and neighboring skin, slight but distinct protrusion of the eyeball, and swelling of the retinal veins; T., S., and F. normal. These symptoms diminished later, were almost gone the next day, but returned more markedly on the following days; abated, and reappeared again. Convinced that the case would follow the same course as the similar ones on record, I told the

[1] *Traité*, tome ii., p. 379.

patient and his father—both extremely sensible men—that in my judgment the other eye also was threatened with blindness, and only an operation could possibly prevent it. The operation, however, would not be without danger. Both father and son unhesitatingly said: "We are willing to take any risk to avert blindness."

I went to Dr. Frank Hartley, one of the attending surgeons of the New York Hospital, of whom I knew that he had several times extirpated the Gasserian ganglion, and asked him whether he was willing to clean out a cavernous sinus (which I supposed to be obliterated by a non-infective thrombus), and prevent the thrombus from extending to the other, by ligating or compressing with a tampon the circular sinus. He seemed startled at first, but when I had told him the history of the case and he had examined the incident literature, and we had discussed the case together a second time, being convinced that total blindness and subsequent death would be inevitable, he said: "I will do it." The greatest counter-indication was the possibility of the presence of a traumatic sarcoma, but this we could not yet assume with any degree of certainty.

The patient was transferred to the New York Hospital February 28th, and operated on there March 1st by Dr. Hartley in the presence of a number of physicians, myself included.

Dr. Hartley, on my invitation, was kind enough to be present at the meeting of the American Ophthalmological Society, at which he described the **operation** as follows:

"The patient was prepared in the usual manner for operation. The anæsthetic used was nitrous oxide and ether.

"An incision was made over the temporal region (Fig. 2.)—horseshoe in shape—and situated as in the operation for the extirpation of the Gasserian ganglion. The periosteum was divided in the line of the skin incision. The bone was divided by means of Doyen's drill, Obalinski's dural depressors, and Gigli's wire saw, and in shape similar to an equilateral pentagon. The osteoplastic flap thus formed was raised and reflected over the zygoma. The dura mater was loosened from the squamous and pourest portions of the temporal bone sufficiently to recognize the second and third divisions of the fifth nerve and the middle meningeal artery at its entrance into the middle fossa of the skull. The middle meningeal artery was now divided. Its distal end was managed by compression beneath the brain retractor, while its proximal end was plugged with catgut in the foramen for its passage through the sphenoid bone.

FIG. 2.

"At this stage the dura mater was incised just above the cavity of Meckel, enclosing the Gasserian ganglion, which, being followed by the escape of cerebro-spinal fluid, allowed a greater field for the operative work. This escape of fluid allowed a very good field for work, but it implied that the retraction of the brain must be carefully and evenly carried out. The cavernous sinus was now located. In front of the junction of the second and third divisions of the fifth nerve the cavernous sinus was incised longitudinally as far as the sphenoidal fissure. No hemorrhage took place at this time, since the vein lumen was occupied by a clot, which filled it, and which was removed without difficulty, but not in its entirety. In incising this portion of the sinus wall, the division of the third and fourth nerves and the ophthalmic branch of the fifth was not thought to be of moment, since they had already shown evidence of paralysis.

"After the removal of the clot in this portion of the sinus a fine probe was passed into the sinus beneath the fifth nerve and toward the superior petrosal sinus. When this point was located, the incision in the cavernous sinus was extended and the remaining clot removed. The entrance of the superior petrosal sinus, which at first did not bleed, was seen.

"After assuring one's self that no portion of the clot remained in the cavernous sinus, the circular and the superior petrosal sinus were investigated carefully with a fine probe. From these sinuses blood soon flowed, but from the ophthalmic vein no blood escaped. The escape of blood from these two sinuses was thought to be sufficient and satisfactory to demonstrate their permeability. The walls of the cavernous sinus were now allowed to drop together, and, as they opposed very accurately, a small strip of sterile gauze was placed against them and over the entrance of the circular sinus. The end of this piece of gauze was brought out through the lower angle of the wound in the skull. This gauze was small in amount but very accurately applied over the sinus. The brain was now allowed to fill the fossa, which it did in about twenty minutes. There was no hemorrhage at this time. The osteoplastic flap was replaced and sutured, but in such a manner that the gauze could be easily removed on the third day without greatly disturbing the flap.

"The condition of the patient at the conclusion of the operation was good. He left the operating room in excellent condition."

The **subsequent history** of the case is as follows:

There was no affection of the right eye; he could always see

well with it, and its outer aspect and the background remained normal. The left eyeball has sloughed, the tumor has continued growing steadily: at the margin it is nodular; it has encroached on the temporal side (Fig. 3), the region of the brow, and the glabella (Fig. 4). At the upper nasal corner it begins to slough (Fig. 3). The diagnosis of a traumatic sarcoma is evident, and as, according to general opinion, with which my own experience agrees, these tumors are never cured, and operations hasten rather than retard the fatal issue, I did not advise any further operative interference.

The course after the operation was almost free from fever during the first three weeks. When the tumor began to ulcerate, the temperature rose, varying between 100° and 103°. The pulse, accelerated during the first days, sank from 124 on the day after the operation to 80 and 72 in the next four days.

In the right eye nothing except swelling of the superior retinal vein was noticed during the first two weeks. Slight haziness of the background disappeared gradually so that the interior of the eye from the third day on could be pronounced normal. S., T., the aspect of the iris, and the behavior of the pupil showed no change during the whole course of the disease. The tumor grew steadily and assumed more and more the character of sarcoma. It extended beyond the superior orbital edge as well as over the temple, the root of the nose, and the glabella (see figures 3 and 4). Toward the temple its surface was uneven and nodular. The inner portion, which had been less consistent than the remainder from the beginning, showed in the third week softening and decay, steadily extending in breadth and depth and smelling offensive. The odor was mitigated by a moist dressing. The patient became more and more apathetic, his hearing decreased, he was drowsy and weak, and **died comatose** May 16, 1900. His vision remained good as long as he was conscious.

The **autopsy** was made in my presence by Dr. G. P. Briggs, the pathologist of the hospital, the day after death. Of his report I shall use what is of importance for our purpose. "The upper half of the nose, the left eye, the orbital tissue, and the adjacent soft parts of the face present a gray, necrotic mass, surrounded by an indurated thick margin, particularly marked on the temporal side. On section, the neoplasm looks more inflammatory than sarcomatous. The piece of bone temporarily resected by Hartley's operation is fastened by connective tissue in its

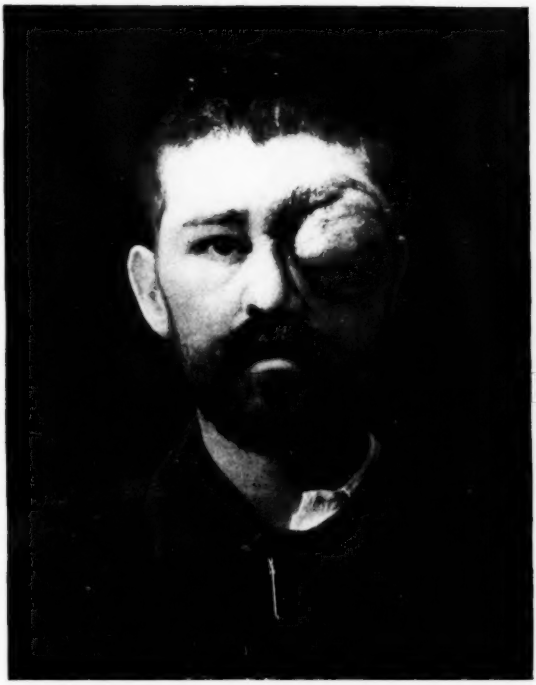

FIG. 3.

FIG. 4.

proper position corresponding to the field of operation, the dura is moderately thickened and slightly adherent to the skull. After detaching the anterior half of the left frontal lobe, a layer of cheesy material, like inspissated pus, of about 1½ *mm* in thickness, is noticed on both sides of the greatly thickened dura. The thickening of the dura extends beyond the cribriform plate, and in part over the left temporal lobe. Otherwise the meninges are healthy. All the sinuses are normal except the cavernous, which cannot be demonstrated. The anterior surface of the right petrous bone and the adjacent portion of the squamous and mastoid portions are smooth but blackish, especially the roof of the tympanum. On opening, the whole interior of the right petrous bone was found uniformly filled with a slightly greenish, caseous material, while the surface of the bone is intact. The left petrous bone showed a circumscribed superficial corrosion on the lateral part of its anterior surface, whereas its interior structure is normal. The roof of the orbit and the cribriform plate are much corroded. The sphenoidal, ethmoidal, and frontal sinuses are filled with ropy matter. The convolutions of the left hemisphere are somewhat flattened. The left oculomotor and the left optic nerve, together with the left side of the chiasm and the adjacent left tract, are thickened. The lymph nodes of the left cervical region are enlarged. They constitute a mass of 5 x 2.5 *cm.*"

The whole tumor, the unopened brain with the dura, and the two petrous bones were given to me and put at once for the night in a 5 %, then in a weaker, solution of formol.

I desire to add to this report the following observations based on the results of the examination of the hardened specimen. The tumor was found to be, at the place of its origin, the orbital roof, as in its whole quite large extent, a small- roundcelled sarcoma, which had softened and destroyed a part of the tissues it invaded. The orbital tissue was converted into a uniform sarcomatous mass, at the apex of which the shrunken eyeball was easily recognized by preserved remnants of lens, choroid, and sclerotic. The skin and subcutaneous tissue, with the exception of the epidermis, had been destroyed by the neoplasm. The bone in the anterior and horizontal portions of the frontal had mostly disappeared; in the farther portions it was corroded.

The inner surface of the thickened dura mater, covering the orbital roof, presented an impression of the upper surface of the orbital roof. The outer side of this part of the dura mater and of the adjacent portions of the frontal lobe of the brain showed the same aspect. Under the microscope this mass consisted of an accumulation of more or less well-preserved, small, nucleated round-cells, which, in the harder portions of the tumor, appeared uniform and dense, with a very scant fibrillar matrix, in the softer parts irregular, lymphoid, and shrunken.

The dura was thickened by infiltration and covered on both its surfaces with spherical and conical sarcomatous proliferations. The fibrous structure of the dura was preserved and pervaded by the small sarcoma cells in quite the same manner as we see it so often at the sclera in small-celled melano-sarcoma. The surfaces of the corroded bone could be cut so as clearly to show the invasion of the sarcoma cells in passages, clusters, and diffusely, substituting the osseous tissue. Suppuration was nowhere noticed; the whole was cell proliferation and destruction of infiltrated tissue through molecular softening and decay. The presence of muco-pus in the accessory cavities of the orbit should not astonish us, for it is a constant companion of the malignant new formations which perforate the bony wall.

The **behavior of the chiasm** was remarkable. The proliferation of sarcoma cells had invaded the left optic nerve from its beginning so that it could not be seen in the orbit. The sarcoma after its perforation of the left orbital roof had extended to the cribriform plate, the adjacent bone and dura, and the contiguous cerebral substance up to, but no farther than, the entrance of the right optic nerve into its canal. Here a very peculiar picture was seen (see Fig. 5): the left optic nerve (*s*) was considerably thickened (8 *mm* in diameter, the right (*d*) being 4.5 *mm*), its color was reddish and its surface uneven, while the right optic nerve was uniformly smooth

FIG. 5.

and white, and continued its course unchanged up to the lateral side of the chiasm. From there on the *lateral* half (*l*) of the right optic tract preserved the appearance of the right optic nerve, whereas the *medial* half (*m*) of the right tract was reddish and distinguished by a sharp line from the lateral half. Not only the left optic nerve but also the left half of the chiasm (*ch*) and the left optic tract were broader and darker (reddish) than the corresponding half on the right side. To judge from appearance, the lateral fascicle of the right optic nerve continued its course strictly in its lateral direction, and the fascicle (*m*) which joined it on the left side of the chiasm on its way farther back was, apparently, derived from the left optic nerve. It was only slightly broader but a little more colored than the uncrossed fascicle (*l*) of the right nerve. At the left tractus no such differences could be made out by the naked eye. The picture seemed to furnish a conclusive example of the semi-decussation. Loath to spin out this article too long, I examined the chiasm and the two nerves only cursorily; the left was pervaded by sarcoma cells. The beginning of the right optic nerve, which was situated at the limit of the extension of the pseudoplasm, likewise contained sarcoma cells, but not in great numbers. The specimen appeared macroscopically too valuable to destroy it by sections.

CONCLUDING REMARKS.

I. In regard to the *diagnosis* there was at first, I think, a pardonable uncertainty. The patient mentioned a blow with a billiard cue to the lid and upper orbital margin, but did not feel any inconvenience from it during the first two weeks; then the upper lid swelled, became evenly red, and hung inert over the protruding movable and well-seeing eye. Then conjunctival œdema, exophthalmos, stasis of the retinal veins, neuro-retinitis, progressive impairment of motion up to complete stiffness of the eyeball, gradual diminution of sight, keratitis from exposure of the cornea, shrinkage of the eyeball, and insensibility in the region supplied by the first division of the fifth pair, made their appearance without

symptoms of fever. Locally there was neither spontaneous nor pressure pain. The temporal part of the dark red uniform swelling of the lid was hard, the nasal part soft. Consulting general and ophthalmic surgeons, among others Dr. Meding, who had sent the patient to the clinic, shared with me the uncertainty of the diagnosis, but thought that a traumatic extravasation of the orbit was the most probable cause, and recommended an exploratory incision in the upper-outer part of the orbit to relieve the tension of the tissues. I did not agree with this proposition, because a possible simple fracture might have been converted into a complicated one. Soon the formerly soft medial part of the lid-tumor began to become still softer, almost to fluctuate. An exploratory puncture let out a small quantity of bloody serum. The diagnosis was not made certain by the puncture, but made it incline toward a traumatic sarcoma. I remembered two cases in children who showed the same symptoms after a blow and both died of sarcoma of the lids and orbit. In the one, the centre of the lid-tumor was softer, redder, and showed a rhythmical bruit on auscultation, which impressed on my mind the diagnosis of a traumatic, palpebral aneurism. I took the child to one of our most experienced surgeons, Dr. W. McBURNEY, at the Roosevelt Hospital. He said: "That looks like aneurism, and on account of this probability I shall try to expose it, but the possibility of a traumatic sarcoma cannot be excluded." I was present at the operation. The place where the bruit was heard was a focus of softening in a lardaceous sarcoma. As soon as Dr. McBurney saw this he desisted from the operation. The child died in about two months. A very similar case without aneurismal bruit was brought to me by the mother of the diseased child.

The partial softening of sarcomas easily leads us to mistake these tumors for cysts. Such a case I communicated to the Ninth International Ophth. Congress at Utrecht in Aug., 1899.[1] The tumor was situated above the inner cauthal ligament and presented itself, after eversion of the

[1] H. Knapp, *Trans. Ninth Internat. Ophthal. Cong.*, Utrecht, 1899. "Some Rarer Orbital Tumors. Five Cases."

upper lid, as a very soft, bluish intumescence which I took to be a serous cyst, extirpated it unbroken, put it in formol, and when I examined it later on was astonished to find it uniformly hardened. It was a small-celled sarcoma surrounded by a thin connective-tissue capsule.

Concerning the uncertainty of diagnosis I may be allowed to cite two places of the excellent *Traité des Maladies des Yeux* of Panas[1]: "Certains sarcomes sont fluctuants, et une fois ponctionés ils laissent écouler du liquide sanguinolent ou citrin d' où la possibilité de les confondre avec des kystes.

"Comme souvent un traumatisme précède l'évolution du néoplasme et que parfois l'une ou l'autre paupière devient spontanément écchymotique, on peut penser à tort qu'il s'agit d'hématome."

II. With regard to the *indications* and the *prognosis* I refer to what I have said in the history of the disease, page 464, under "Extended Clinical Diagnosis."

III. The *prognosis* suggests some further scientific and practical remarks. At the post-mortem examination, the thrombosis of the cavernous sinus could not be demonstrated, for the sinus had disappeared in the process of destruction by the progressing pseudoplasm. During the operation, which I watched very carefully, the sinus presented itself in the same manner as we see it in operations on the mastoid, when it is totally or partially occupied by a non-infected thrombus. The sinus, slit open, showed an uneven calibre, in the midst of which there were two oval black thrombi, which could be easily removed, whereas on the sides the sinus wall was thickened by dull-yellow, stratified, non-removable deposits — organized thrombus.

The thrombus was aseptic, for during life the symptoms of pyæmia and at the post-mortem examination the conditions produced by septic thrombosis — purulent disintegration of the contents and walls of the sinus, serous and purulent meningitis—were absent. Sometimes we have an opportunity to see with the ophthalmoscope a miniature picture of

[1] Panas, *Traité*, tome ii., p. 445.

aseptic coagulations in the veins of the retina; by exact adjustment in the upright image we notice an oval black deposit, immovably adherent to one side of a vein, whereas between the deposit and the opposite wall of the vein there is a small, brighter interval which I take to be flowing blood.

On both sides of the obstructed cavernous sinus there were obturating thrombi, namely, in the circular and superior petrosal sinuses. A blood current could be produced only by passing probes into these two sinuses, an observation with which aurists are quite familiar in septic thrombosis of the sigmoid sinus. The rapid, almost sudden, appearance of the symptoms of thrombosis, from one cavernous sinus to the other, which showed itself so marked in our patient, is also mentioned by Panas.[1] "Un charactère à peu près constant de la thrombose primitive du sinus est la bilatéralité de l'exophthalmie qui se produit en quelques heures, ou d'un jour á l'autre, au milieu d'accidents cérébraux graves." The presence of grave cerebral symptoms was, however, not noticed in our case, but merely ocular symptoms, and these were not constant, but intermittent during the seven days between their first appearance and the operation. Evidently transient and incomplete thromboses developed, and during the last days the grave symptoms were almost altogether wanting, probably because by the formation of obturating thrombi in the circular sinus, which were demonstrated during the operation, the blood current from the cavernous sinus and the orbito-ocular veins was no longer interfered with from the diseased side, constituting an intermission frequently noticed in sinus thrombosis.

In its kind the tumor was found to be a *small-celled sarcoma* with infiltration and extensive destruction in the neighboring tissue. The roof of the orbit was covered on both sides with a layer of a white, soft substance; the uneven thickness and small-pitted surface gave it an exquisitely corroded aspect. The grumous deposits were slightly moist, resembling necrosed tissue rather than inspissated pus. I

[1] *Traité*, tome ii., p. 379.

have sufficiently dwelt upon the propagation of the sarcoma to the neighboring parts, and the changes produced by it. Whether the sarcoma was really produced by the blow of a billiard cue I can of course not demonstrate, but there was no other assignable cause, nor the presence of sarcoma in any other part of the body.

IV. The presence of an *aseptic thrombosis* in the cavernous sinus was a *matter of direct observation* for me and all that were near enough during the operation. If it had been caused by a chronic suppuration of the middle ear we would have had to deal with an entirely different picture of disease and autopsy. The above-mentioned symptoms would have been accentuated by those of pyæmia: chills, steep-pointed pulse-charts, pus in the sigmoid and petrosal sinuses, purulent meningitis. Nothing of the kind was to be noticed. What was the true or at least the most probable cause of the thrombosis, the injury or the sarcoma? Either may have been the cause. The most probable supposition seems to be that the injury caused the sarcoma, and the sarcoma by pressure on the orbital tissues caused the thrombosis of the ophthalmic vein and the cavernous sinus.

V. What *practical applications* may be derived from the above observation for the diagnosis, prognosis, and treatment of similar cases? For the diagnosis, the case has the value which an important, unusual, and complete observation always possesses. The prognosis of the traumatic sarcoma of the orbit is hopeless in general. I do not believe that we could have saved the patient if we had made the correct diagnosis at the beginning. For *treatment* the case is eminently *valuable*, for Dr. Hartley's skill has demonstrated how the sinuses in the anterior part of the base of the skull may be reached. The method of osteoplastic resection, in this almost inaccessible neighborhood, simultaneously practised and made known by Hartley and Krause, may be applicable not only in the rare cases of aseptic thromboses of the cavernous sinus, but also on the infective otogenous thromboses.

SPASMODIC ACTION OF THE OBLIQUES IN CASES OF ABDUCENS PARALYSIS.

By ALEXANDER DUANE, M.D., New York.

IN the May number of the ARCHIVES OF OPHTHALMOLOGY, Dr. Wolff has described an interesting case in which with paralysis of the external rectus there were retraction movements of the affected eye. As Dr. Wolff says, this same case had already been reported upon by Kunn, who, apart from the retraction movements, gave a very accurate and elaborate description of it.[1]

It so happens that I myself have examined this very case on three separate occasions, and, while my findings essentially agree with those of the two observers just cited, it has seemed to me that the significance of one feature that it presented has escaped them both. I refer to a spasmodic action of the inferior oblique, producing a marked upward deflection of the eye when rotated inward. I have observed a similar action in two other cases, in both of which there was also a paralysis of the external rectus. In view of the peculiarity of the conditions presented, a report of them may be of interest.

CASE I. **Congenital Paralysis[2] of Left Externus with moderate Paresis of Left Internus (particularly marked in binocular vision). Spasm of Left Inferior Oblique.**—Louise M., aged nineteen. Conditions noted have existed since early childhood, and probably since birth. Interior and vision normal.

[1] *Beitr. zur Augenheilk.*, xix., 1895.

[2] For the sake of brevity, the word "paralysis" is used to denote insuperable restriction of movement, whether due to defective innervation, or to non-development of the muscular fibre, or to any other cause.

When attempts are made to perform associated parallel movements with both eyes uncovered, the right eye apparently moves normally in all directions, although extreme movements, especially outward, are performed with effort, and, in the case of the outward excursion, are attended with nystagmus-like oscillations. In the horizontal plane, **left eye** can move outward only as far as the middle line or a short but varying distance beyond it. In the lower field, it can move outward rather more, and *in the upper field more still*, so as to perform quite a degree of excursion up and out. Movements of left eye up and down normal, but *as eyes ascend the left eye turns out*, the divergence being pronounced both when the patient looks straight up and also when she looks up and to the right or up and to the left. Excursion of left eye inward deficient, inner edge of cornea just touching caruncle. As left eye is carried inward, *it shoots obliquely upward*, and at the same time lids contract so that cornea is half buried beneath the upper lid. This oblique upward movement, already pronounced in the horizontal plane, *i. e.*, when the attempt is made to carry the eyes straight inward, *becomes still greater above the horizontal plane, i. e.*, when the eyes are directed upward as well as inward, and on the contrary *diminishes markedly when the eyes are directed downward.* It is equally apparent if the contraction of the orbicularis is opposed by lifting the left eyelid with the finger while the patient rotates the eye in. This upward deflection, while marked in all three examinations, evidently varies in amount from one time to another in the same way that the diplopia does (see below).

In the left half of field, there is homonymous diplopia, beginning in the primary position (sometimes a little to the right of it, sometimes a little to the left) and increasing fast as the eyes are carried to the left. When looking up and to the left, there is single vision, giving place to homonymous diplopia as the eyes are carried still farther to the left. The same is true for downward directions of the gaze, except that here homonymous diplopia begins to be apparent sooner as the eyes are carried to the left. No vertical diplopia in the left half of the field of fixation.

In right half of field of fixation, crossed diplopia beginning at from 5° to 10° from the primary position, and increasing fast as eyes are carried to right. *Vertical diplopia* with the image of the left eye below (left diplopia), *increasing as the eyes are carried to the right*, and becoming great when eyes are carried *up and to the right.* When eyes are directed to the right and then are carried

down, the vertical diplopia *diminishes as the eyes fall below the horizontal plane*, and ultimately becomes zero. The amount of vertical diplopia varies at different examinations, but is always marked, the vertical distance between the images in the upper left-hand field being as much as 25 centimetres when the test object is 1 metre from the eyes, representing an actual deviation of 10–12° (= 23° of prism).

The range of movement of each eye singly is shown in the accompanying measurements of the field of fixation, taken with the perimeter and double dot.[1]

FIELD OF FIXATION.

	June 15.		July 16.
	Right Eye.	Left Eye.	Left Eye.
Out	68°	20°	20°
Out and 30° up			25°
" " 45° up	55°	15°	
" " 60° up			30°
" " 75° up		46°	40°
Up	38°	36°–42°	40°
Up and 15° in		46°	40°
" " 30° in			40°–48°
" " 45° in	48°	35°	
" " 60° in			44°
In	56°	35°	42°
In and 30° down			40°
" " 45° down	45°	38°	
" " 60° down			48°
Down	68°	58°	69°
Down and 30° out			30°
" " 45° out	70°	24°	
" " 60° out			20°

These measurements indicate that in *monocular fixation* there is great restriction of the movement of the left eye outward, moderate restriction of movement inward, and restriction of movement of both eyes downward and inward. Movements of left eye upward normal,[2] and, if anything, increased upward and inward.

[1] This test, which seems to combine precision with accuracy, is made by fixing the patient's head firmly in such a way that the eye examined occupies the centre of the perimetric arc and when in the primary position is directed at the zero of the scale. Keeping it in this position, the examiner carries along the perimeter arc a card having upon it two fine dots set close together. The patient follows the card with his eye without moving his head. The moment that he fails to follow the card, the two dots, being no longer sharply fixed by the eye, will appear in diffusion circles and hence will blur into one. Therefore, all that we have to do in order to ascertain the limit of excursion in any one direction is to find at what point of the arc the two dots can no longer be seen separate but appear to run into one.

[2] Upon what grounds Kunn (*l. c.*, p. 157) diagnosticated a paresis of the left elevators in this case, I am at a loss to say. There is absolutely no evidence of such a paresis now.

In performing *convergence with both eyes uncovered*, the right eye fixes and the left diverges; the near point of convergence being about thirty-two centimetres from nose when the eyes are directed straight ahead, twenty-five centimetres if the eyes are directed 5° to the left, and sixteen if the eyes are directed 10° to the left (insufficiency of action of left internus).

If *convergence is performed with the right eye covered*, left eye can turn in upon object of fixation until the latter is not more than five centimetres from the nose and the inner edge of the left cornea is considerably inside of the caruncle.

It is thus apparent that both for parallel and convergent movements the left eye moves much *better when the right eye is excluded* than it does in binocular vision. The same is true to a less extent of the right eye.

Here there was evidently a nearly complete paralysis or, more properly speaking, inaction of the external rectus. This inaction, which Kunn with reason regards as congenital, was probably structural, being due to non-development of the externus itself. There was also a marked insufficiency of the internus. This was much more pronounced for binocular than for monocular vision and is hence to be regarded as a paralysis of movement[1] rather than a paralysis of the muscle, *i. e.*, is due to an affection of one of the association centres and not to any impairment either of the muscle itself or its supplying nerve.[2] In addition there were the peculiar behavior of the orbicularis and the peculiar retraction of the eyeball, to which Wolff has called attention, but which, I may say, were little pronounced when I examined the case.

For the *marked upward deflection* occurring in the left eye whenever it was turned inward, Kunn offers no explanation. Wolff suggests that it is due to the resistance offered by the optic nerve to the backward movement of the eye as the latter is retracted into the orbit—the eye being, so to speak, squeezed between two opposing forces, which, because they

[1] Blicklähmung (Kunn). (Fixation paralysis.)

[2] Wolff's theory that the restriction in adduction was purely mechanical does not seem compatible with the fact that this restriction was less marked for monocular vision than it was when both eyes were used, and in the latter case was much greater for convergence than it was for associated parallel movements. If purely mechanical it ought to have been the same under all conditions.

are not precisely in line with each other, tend to thrust it upward.

That this explanation is at all adequate I cannot think. The resistance offered by a flexible body like the optic nerve must be slight indeed and could scarcely be constant in character and direction. The deviation due to it, therefore, would be irregular and subject to sudden variations, and not marked by the uniform steady increase in the upper field and the uniform steady decrease in the lower field shown in the present case.

The vertical deflection might be ascribed to an anomalous insertion of the internus, causing it to act partly as an elevator; but a similar objection to that made against Wolff's hypothesis applies here—namely, that a deviation due to this cause would not be essentially greater when the eyes were directed up than when they were directed down.

Furthermore, neither theory accounts at all for the singular fact that the left eye, in spite of the almost complete inaction of its externus, diverges markedly and progressively as the eyes are elevated, and when elevated can be abducted to quite a considerable degree.

On the other hand, all the conditions are explained upon the hypothesis of an *excessive spasmodic contraction of the inferior oblique.* Such a contraction would tend to carry the eye upward, and the more so in proportion as the eye is turned inward. The ability of the inferior oblique to carry the eye upward, moreover, would increase steadily in the upper field of fixation and diminish steadily in the lower field. Again, when the eye was carried upward, and especially when it was carried upward and outward, the abducting action of the inferior oblique would come into play, and if excessive would cause the eye to diverge in spite of the coexisting paralysis of the externus.[1] Furthermore, in upward and especially in upward and outward directions of the gaze, the abducting action of the inferior oblique, if excessively

[1] The divergence observed in the left eye when looking up and to the right cannot be ascribed to this spasmodic action of the inferior oblique, since this muscle does not act as an abductor when the eye is directed up and in. It is evidently due to the simultaneously existing insufficiency of the left internus. The divergence of the left eye in looking down and to the left is probably due to a similar but slighter action of the superior oblique.

exerted, would enable the eye, as in this case, to move outward moderately even in default of any action of the externus.

The character of the diplopia and the way in which it increases and decreases, as also the manner in which the field of fixation is restricted, are strictly in accord with this hypothesis.

The fact that the diplopia and deviation varied in amount from time to time while always preserving the same character lends probability to the assumption of its spasmodic origin.

It may be added that A. Graefe [1] has cited an instance of a complete paralysis of the externus in which the obliques acting alone were able to produce a moderate amount of abduction of the eye when the latter was directed up and down, but not, of course, when directed horizontally. In a case of incomplete paralysis of the externus, in which the eye could be abducted somewhat while still in the primary position, this compensatory action of the obliques would come much more markedly into play, since the abducting power of the obliques increases with the degree of abduction of the eye.

An altogether similar case is that of the sister of the foregoing, likewise cited by Kunn [2] and Wolff.[3]

Also similar is a case given by A. Graefe [4]:

> Eight-year-old boy with complete paralysis of the left externus existing since infancy. If the object of fixation is carried horizontally to the right, and both eyes are made to follow it, there is marked contraction of the left palpebral fissure, while *if the object is carried up and to the right the left eye moves upward excessively.*

And Kunn [5] gives a case which, while not precisely parallel to those cited, yet is similar in that it is apparently a case of spasm of both elevators (principally the inferior oblique) of the left eye:

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[1] Graefe-Saemisch, *Handbuch d. ges. Augenheilk.*, 2d ed., Bd. viii., Kap. xi., p. 88, Case 13.
[2] *L. c.*, p. 63.
[3] *L. c.*, p. 300, Case 4.
[4] *L. c.*, p. 87, Case 6.
[5] *L. c.*, p. 68.

Seventeen-year-old boy. When the two eyes are examined separately, the eye not under examination being kept closed, the abduction and adduction are found to be limited in the right eye, and absolutely *nil* in the left—*i. e.*, with the right eye closed, the left eye remains perfectly motionless when the attempt is made to get it to follow an object carried to either the right or the left. But when the same attempt is made with both eyes open, and the eyes are directed to the right, the *left eye flies straight upward* until nearly the whole pupil is buried beneath the upper lid. The left palpebral fissure at the same time contracts and the patient wrinkles his forehead. In looking up, both eyes move well, but the *left moves up better* than the right. In looking down, on the contrary, the right moves better.

My own cases in addition to the one cited are the following, which have been elsewhere reported[1]:

Case 2. **Paralysis of Left Externus; Spasm of Left Inferior Oblique.** Bertha S., aged eight. Deviation of eyes noticed since birth. Used to hold head to right and does so still when looking intently at anything. Left eye cannot move out beyond median line. *When eyes are carried horizontally to the right, left eye suddenly flies upward* and becomes buried beneath the upper lid. *If the eyes are directed down and then carried to the right, left eye does not fly up*, but moves on same plane as the right. Behind screen, left eye deviates high up and somewhat in. No double images attainable.

Case 3. **Paralysis of Right Externus; Spasm of Right Superior Oblique.** Emma D., aged fifteen. Strabismus since scarlatina eleven years before. Occasional vertigo; no diplopia. Right eye deviated inward, and when screened moves downward. Movements of left eye normal. When both eyes are directed to the right, right eye cannot move out beyond middle line; nor does it move up at all when the eyes are directed up and to the right. Movement of right eye almost normal when eyes are directed up and to the left (retention of power of the inferior oblique); movement downward excessive, especially when eyes are directed downward and to the left (excessive action of superior oblique). When attempt is made to move eyes straight to left, right eye shoots obliquely down and to the left (spasmodic action of the superior oblique).

[1] *Motor Anomalies of Eye*, pp. 54, 55.

That the downward deviation of the right eye in this case was due to the overaction of the superior oblique and not of the inferior rectus is evident from its marked increase when the eye is turned inward — a direction of the gaze in which the inferior rectus has little power as a depressor.

Not associated with a paralysis of the externus and hence not strictly analogous to these cases but still of kindred nature, is the case I have reported,[1] in which to the ordinary evidences of a paralysis of the left superior oblique there were, after a time, superadded symptoms of spasm of the left inferior oblique — *i. e.*, in movements up and to the right the left eye rose higher than the right, and when the attempt was made to carry both eyes straight to the right, the left eye shot obliquely up.

The overaction of the obliques in the cases cited is probably *compensatory in character* — *i. e.*, the abducting action of the obliques is called into play to an excessive degree to replace as well as may be the defective abducting action of the externus. There is, however, another possibility. In marked retraction of the eyeball, the tendons of the obliques will be put on the stretch, and the tendons of the recti will be relaxed. If the retraction takes place to the extent that Wolff believes, the effect of the obliques may be so heightened by the extra tension, and the effect of the recti so diminished by their relaxation, that a relative excess of action of the obliques may be developed. This theory, however, hardly accords with the facts in Case I, in which the overaction of the inferior oblique was evident in directions of the gaze (up and also up and to the left) in which there was no retraction of the eyeball. Moreover, the absolute amount of retraction shown by the case when I had it under observation was slight, and, I am inclined to think, more apparent than real—the appearance of refraction being simulated by the contraction of the orbicularis.

It may be added that the existence of a vicarious action of the obliques producing a certain amount of abduction even in complete abducens paralysis has long been noted—*e. g.*, by J. Soelberg Wells,[2] and also by Streatfeild,[3] although the

[1] Archives of Ophthalmology, xxvi., 3 (1897), p. 325.
[2] *Ophthalmic Hospital Reports, II.* (1859). [3] *Ibid.*

latter gave an erroneous interpretation to the phenomenon. Such a vicarious action is shown by spasmodic outward movements of the paralyzed eye somewhat beyond the middle line, the eye at the same time moving in a zig-zag direction up and down. This action is of the same nature as that presented in the cases here cited, differing, however, in being transient in character and but moderate in degree.

THE CHROMOSCOPE: A CONVENIENT INSTRUMENT FOR STUDYING THE COLOR SENSE OF THE MACULA LUTEA AND ITS ANOMALIES.

By Prof. M. KNIES, Freiburg-in-Baden.

(*With three figures in the text.*)

Translated by Dr. Ward A. Holden.

WHILE studying certain anomalies of the color sense it seemed to be advisable to determine the limits of the various colors in the spectrum as seen by persons with normal and abnormal color-perception. An examination of this sort is usually considered unreliable. It was soon discovered, however, that the variations when a single individual was tested, myself for example, were very slight. With different individuals, even when their color perception is perfect, the statements as to the limits between the different colors vary much more, within certain bounds indeed, but not such narrow ones that the variation can be supposed to depend upon errors of observation alone. Corresponding to this we find that the limits of the various colors in the spectrum have been differently stated in the literature where theoretical considerations have also come into play.

I began with experiments on myself, using a Bunsen spectroscope and an Auer burner. The first purpose was to determine the limits of the spectrum as regards the perception of color only. Luminosity and width of slit have their influence, but for the comparison of various eyes I employed an arrangement with which the red end of my spectrum fell at 173 of the arbitrary scale (the sodium line D being taken as 200) and the violet end at 293. For my own eye red has its

greatest intensity at 189.5 and violet at 276. The boundary between reddish yellow and greenish yellow (neutral yellow) lies at 202; that between yellowish green and bluish green (neutral green) at 215.5; that between greenish blue and pure blue (sea blue) at 228. In Fig. 1 these results are given in a graphic manner. The markings represent the average of half a dozen observations, the variations, however, being very slight.

The division into colors is of course quite different if we transpose the arbitrary scale of the spectroscope into wave-lengths. In Fig. 1, above the horizontal line are given the spectrum lines, with the help of which this transposition was made. We obtain then the following wave-lengths expressed in millionths of a millimetre:

1. Outer boundary of red	742
2. Greatest intensity of red	641
3. Boundary between reddish yellow and greenish yellow	583
4. Boundary between yellowish green and bluish green	538
5. Boundary between greenish blue and pure blue	510
6. Greatest intensity of violet	528
7. Outer boundary of violet	404

In Fig. 2 these relations are given in a graphic manner, and this represents the division of the colors in the spectrum as they appear to over 90 % of all persons with normal vision.

The differences found in different individuals are so slight that when we take into account all sources of error, such as fatigue, the effects of contrast, and the like, the differences easily fall within the limits of errors of observation.

It would be too cumbersome in each case in which the color sense is examined to use a spectroscope. We can determine the more important fixed points in the spectrum (excepting neutral green) in a much simpler manner. When I look at a white strip on a black ground though a prism in such a manner that the well-known colored margins appear, while the middle of the strip remains colorless, I see the following: On one side of the spectral image red begins and, without markedly changing its hue, grows more intense and then passes over rapidly through orange to yellow, which fades

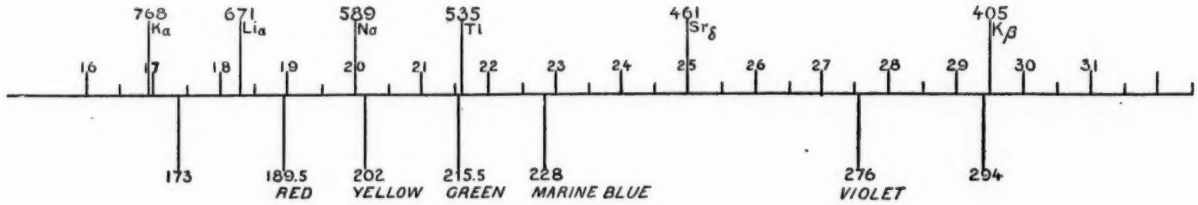

FIG. 1.

Location of the colors in my own spectrum, using the arbitrary scale D = 200; above the scale the spectral lines which served to transpose the limits into wave-lengths.

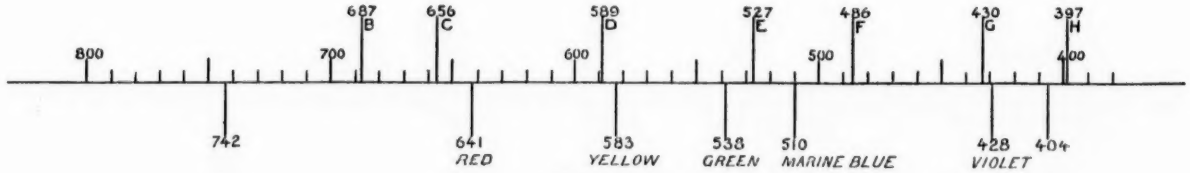

FIG. 2.

Location of the colors in my spectrum in wave-lengths; above the scale the lines B to H of the spectrum.

away into the white of the middle of the strip. On the other side of the strip I see the violet becoming more intense and then passing over rapidly through indigo to marine blue, which fades away to white toward the middle of the white strip. In other words, I see the spectral color λ 641 pass rapidly into λ 583, and the latter into white; while on the other side λ 428 passes rapidly into λ 510 and the latter into white. The spectral hues[1] between λ 583 and 510 are completely wanting.

In this way four principal colors are seen which correspond to the above-mentioned colors indicated as 2, 3, 5, and 6 in the spectrum, and can readily be matched with the colored yarns.

In order to carry out such examinations quickly and conveniently I have constructed a small instrument which I have called a chromoscope. This is essentially an arrangement for exhibiting the colored margins of a white strip without distracting the attention by any other spectral phenomena.

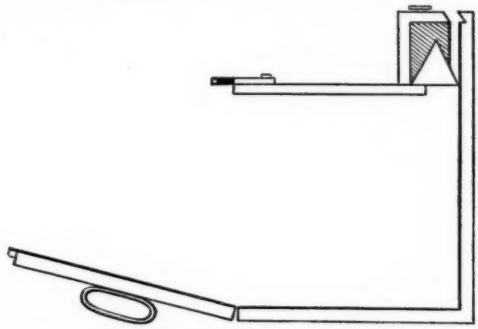

FIG. 3.—THE CHROMOSCOPE. (½ *Natural Size.*)

It consists, as shown in Fig. 3, of a box lined with black velvet, whose front side can be turned down nearly to the horizontal. On the opposite side, above, is a small projection on which is mounted a flint-glass prism having an angle of 60°. A strip of white paper 5–10 *mm* broad is viewed through the prism, one margin or the other being brought

[1] The word "hue" is used throughout in translating the author's term "nuance," in the original, which of course refers to luminosity only and not color, literally meaning tint or shade.—TRANS.

into the field, and the patient is asked to select a skein of yarn similar to the color seen, without calling it by name.

Persons with a normal color-sense select the same hues as I do myself: the end colors of the spectrum, red and violet (any deviation here is abnormal and indicates a shortening of the spectrum), and then a neutral yellow and a marine blue. There may be a little uncertainty above the middle colors, and a greenish blue skein may be selected and even called green.

If now the observer's color sense is abnormal, other colors are seen and other skeins are selected which are characteristic for the sort of color-blindness existing.

What v. Kries found by painstaking examinations and measurements (*Zeitsch. f. Psych. u. Physiol. d. Sinnesorgane*, vol. xiii., p. 241), viz., that there are two sharply differentiated forms of congenital color-blindness: protanopes (red-blind) with a maximum perception in greenish yellow (λ 571) and blue (λ 480–460), and deuteranopes (green-blind) with a maximum perception in orange (λ 603) and blue (λ 480–460), can be discovered with the chromoscope at a glance. The affected person will select at once the proper blue and either yellow or reddish yellow.

It is very interesting to determine with the chromoscope those color anomalies which arise in progressive diseases of the optic nerve and similar processes. In the beginning four colors are chosen here also, but other than the normal colors. The two inner colors of the spectrum, yellow and marine blue or greenish blue, are normally perceived; but instead of red, yellowish red or, later, reddish yellow will be selected, and in place of violet, which is the first color to be lost, the patient selects various hues of indigo up to cyan blue. This indicates a shortening of both ends of the spectrum. So long as four distinctly different colors are recognized in the chromoscope, green will be recognized. Then the condition becomes such that only yellow and blue are seen, just as in congenital color-blindness, except that here the acuteness of vision also has suffered. The two colors seen in the chromoscope become gradually fainter until no color at all is seen in the middle of the spectrum. Actually perception of violet is lost first, then of green, then of red.

The examination with the chromoscope of course only gives us information as to the color perception at the macula. It must be supplemented with color perimetry, in which more attention should be given to tests with violet than has hitherto been done.

Examination with the chromoscope [1] has also led, as the following paper will show, to the discovery and accurate determination of a previously ignored congenital color-defect, a knowledge of which is of great theoretical importance, though its presence leads to but slight clinical symptoms.

[1] The chromoscope may be obtained from Carl Kramer, Friedrich Strasse, 15, Freiburg-in-Baden.

ON A FREQUENT BUT HITHERTO UNRECOGNIZED FORM OF CONGENITAL VIOLET-BLINDNESS AND ON COLOR ANOMALIES IN GENERAL.[1]

By Prof. M. KNIES, Freiburg-in-Baden.

(*With four figures in the text.*)

Translated by Dr. Ward A. Holden.

SOME time ago I described in the *Archiv f. Augenheilk.* (vol. xvii.) an anomaly of color perception in which violet is not seen, while the color sense is normal for the other colors of the spectrum. My report has been almost entirely ignored. It was not reviewed in Michel's *Jahresbericht* for 1887 because "it was not adequate to the present status of color examinations." If this means that it fits neither the Young-Helmholtz nor the Hering color-theory I have no objection to make; nevertheless such cases are not to be done away with simply by ignoring them.

This color anomaly is not particularly rare. Lucanus (*Arch. f. Augenheilk.*, xxi., p. 41), who, to my knowledge, is the only one who has taken up this matter, readily found half a dozen cases which he described. Personally, I regard it as quite as frequent as Helmholtz's red-green blindness or Hering's confounding of red and green. It will seem improbable to many that so pronounced a color anomaly should be so frequent, but here again we find that current theories under certain circumstances are not conducive to the discovery of truth.

This form of color anomaly is congenital and is character-

[1] Criticism of some of the statements in this and the succeeding paper may be found in Raehlmann's paper, *Zeitsch. f. Augenheilk.*, vol. ii.—Trans.

ized by the fact that pure violet is not seen, however intense it may be, while the other colors of the spectrum are promptly recognized and rightly distinguished, excepting greenish blue and marine blue, which the patient has some difficulty in naming. In consequence of the invisibility of violet, red and purple are not distinguished from each other, both being called red. Even a hasty examination with the spectroscope reveals a marked shortening of the blue end of the spectrum. The acuteness of vision, field of vision, and light sense are normal in these cases unless there are complications which would cause functional disturbances even in the normal-sighted.

In ordinary life such persons are regarded as having normal color-perception. It would scarcely be remarked if they called "blue" the violet hues that in nature are rarely pure, for many people do this who have a correct appreciation of violet; nor if they called purple hues "red," as even Hering does in his color system. (The erythropsia or red vision of those who have been operated on for cataract would more correctly be called "purple vision.")

With Helmholtz's theoretical violet-blindness this anomaly, as is readily understood, has nothing to do. Nevertheless we are justified in calling such persons violet-blind, for they are just as blind for violet as the Helmholtz red-blind are blind for red. This violet-blindness is so complete that spectral violet, even in its greatest intensity, is not recognized as a color. There is this great difference, however, that the red- and green-blind see only two colors in this spectrum, while the violet-blind in their spectrum see all the other colors as do persons with normal color-perception, and each portion of their spectrum has a different hue. They must, like those with normal vision, possess at least three color components in order that each point in their spectrum can appear to be of a different hue.

A very pronounced case of this color anomaly which I have recently examined from every view-point furnished the occasion for this paper.

G. M., a physicist of repute, and a personal friend, told me years ago that in astronomical observations he had never

been able to see the violet stars. An examination with the colored wools revealed the defect in question: dark violet = black, purple = red, some uncertainty in the designation of greenish-blue and bluish-green colors, and quick and correct naming and differentiating of all other colors. Tints and shades of any color were properly arranged together, even when the color was purple.

Lately a careful examination was made with the following result:

$$R - 2. \; -0.75c. \; // \; V = \tfrac{5}{6} +.$$
$$L - 2.5 - 0.75c. \; \backslash\backslash \; V = \tfrac{5}{6} +.$$

A slight conus in each eye; optic disc and surrounding fundus otherwise normal.

With the colored yarns the results agreed with those given above. With Stilling's charts, Nos. 2, 3, 4, 6, 7, and 10 were read easily, and Nos. 1, 5, 8, and 9 with somewhat greater difficulty. Ordinarily the examiner in such a case would have reported normal color-perception.

With the chromoscope "red and yellow" were seen at the red end of the spectrum, and "green and blue" at the violet end, and these colors were matched with the yarns, while I myself saw red and yellow at one end, and marine blue and violet at the other. The red selected by G. M. was a yellowish red, a fact to which at first I paid little attention.

For further examination I used a Bunsen spectroscope. I did not purpose to determine the utmost limits of the visible spectrum, but with a bright spectrum to determine the limits of color perception. For these experiments it seemed to me most fitting to arrange the apparatus so that the limit of violet lay at 244 of the arbitrary scale (the sodium line = 200). Under these conditions G. M. found that the limit of the blue end of his spectrum, whose color he called "blue," lay at 281. In Fig. 1, the results are given graphically, where the short lines below the scale refer to G. M., and the long ones to me.

It appeared further, although I had overlooked it before, that there was also a shortening of his red end of the spectrum (176.5 instead of 173). The shortening of the blue end was 13, while that of the red was only 3.5.

Further than this, the following determinations were made:

	KNIES.	G. M.
1. Greatest intensity of red or transition of red to orange	189.5	195
2. Limit between reddish and greenish yellow	202	202
3. Limit between yellowish and bluish green	215.5	214.5
4. Limit between bluish green and pure blue	228	225
5. Greatest intensity of color at blue end (See Fig. 4.)	276 (violet)	258 (blue)

Transposed into wave-lengths and expressed in millionths of a millimetre we find, as is shown graphically in Fig. 2:

	KNIES.	G. M.
Red end of spectrum	742	718
1. Greatest intensity of red	641	623
2. Neutral yellow	583	583
3. Neutral green	538	541
4. Limit between bluish green and blue	510	516
5. Greatest intensity at blue end	428	451
6. Blue end of spectrum	404	421

We found, thus, that under these conditions the red end of G. M.'s spectrum was shortened 24, and the violet end 17, as reckoned in wave-lengths. But on account of the unequal division of the spectrum this color disturbance was chiefly manifested in the blue end of the spectrum. The portion of the spectrum which the normal-sighted name violet, is here almost entirely wanting. Neutral yellow appears at the same point for both of us, but the limit between greenish blue and pure blue is somewhat displaced toward the red end of the spectrum for G. M., thus being explained the fact that his naming of greenish-blue and bluish-green hues was not quite normal. That this error is less marked in the case of red and orange, is due to the fact that the reddish hues of orange, in the lack of a current German name, are usually called merely red. Here I may remark that this confusion is found as a transitory condition in the incipient

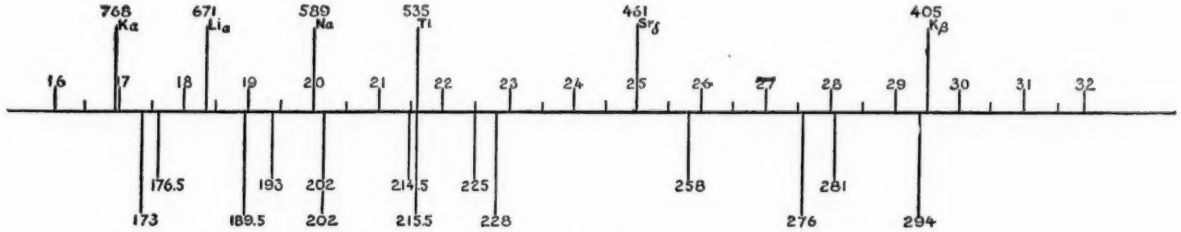

FIG. 1.

Location of colors in the spectrum as seen by G. M. (short lines) and by me (long lines); arbitrary scale (D = 200). Above are the spectral lines used in computing the wave-lengths.

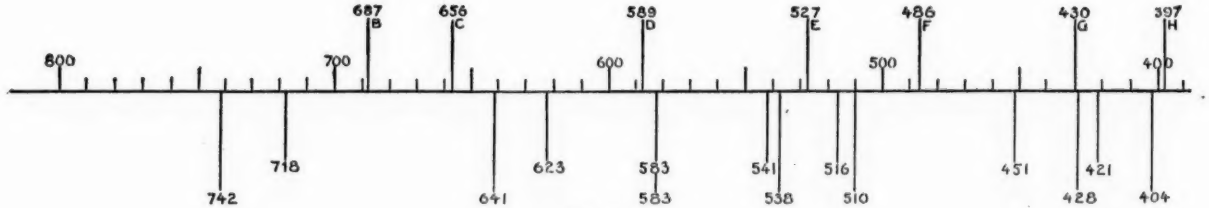

FIG. 2.

Location of colors as above reckoned in wave-lengths. Above the scale are the lines B to H of the spectrum.

stages of optic-nerve atrophy at a time when the acuteness of vision is still normal, red appearing as orange.

There can be no doubt but that the color system of G. M. is made up of other components than mine. The simplest examination of the color system of a person consists in determining accurately the limits of each individual color in the spectrum. For ordinary purposes the chromoscope suffices for this. The person with normal vision will see red (λ 742–641), yellow (λ 583), blue (λ 510), and violet (λ 428–404). The transitions between red and yellow, and between blue and violet, are not altogether wanting, but are inconspicuous. When G. M. was examined with the chromoscope he selected as equivalent colors a reddish orange, yellow, greenish blue, and indigo. These were exactly the colors which we both had called 1, 2, 4, and 5, in the spectrum (see *antea*). The point to be held fast here is that there are persons whose perception of violet is entirely wanting, while they are able to differentiate perfectly all the other colors in the spectrum. Such cases cannot be explained by the three-color theory, for if there be three fundamental color-perceptions, and one of these is proved to be wanting, three cannot remain, yet three are necessary in order that all the remainder of the spectrum be perceived as consisting of different colors.

This form of violet-blindness belongs to that class of facts which, like the processes in optic-nerve atrophy, cannot be made to accord with the Helmholtz theory of color perception. It seems to me in general that the proofs of the three-color theory proceed for the most part from the disproof of the Hering theory, whence, on the supposition "*tertium non datur*," the correctness of the former is supposed to be demonstrated. Thus v. Kries (*Zeitschr. f. Psych. u. Physiol. d. Sinnesorgane*, xiii., p. 241) has proved that Hering's confounders of red and green may be separated into two distinct classes between which are no transition forms, which of course would be impossible according to Hering's theory. But does this prove that the three-color theory is correct? If such color-blind persons possessed only two of Helmholtz's fundamental colors, viz., red, green, and violet, the

protanopes (red-blind) must have green and violet as fundamental perceptions, and the deuteranopes (green-blind) red and violet, called by both yellow and blue. Actually, according to v. Kries, the two forms have an almost exactly corresponding maximum of short-waved perception in blue (λ 480–46), and a maximum of long-waved perception in greenish yellow (λ 571) for the red-blind, and in reddish yellow (λ 603) for the green-blind.

The three fundamental colors can not, therefore, be red, green, and violet, but reddish yellow, greenish yellow, and blue; but these three colors would by no one be made the basis of a color system. However, the fact remains that there are two essentially different forms of the common color-blindness (those who see two colors only) in which the persons confound red and green, which, on the other hand, speaks against the correctness of Hering's theory. In the deuteranope there is wanting not simply a single component of normal color-vision, but the remaining two components differ from the normal. Without going farther, I will leave the matter to the adherents of the two theories to bring them into accord with the facts.

This violet-blindness, I repeat, is congenital, stationary, and not especially rare. In the ordinary tests for colors it escapes our notice, and it must be specially sought for, since a subjective sense of color disturbance is either wanting or very slight. Nevertheless, certain observations may lead us to suspect its presence. I am convinced that Prof. Becker was violet-blind, and it should always awaken suspicion when one calls visual purple "visual red." Perhaps even Hering is not free from this defect.

Lack of sufficient material prevents me at present from determining in how far transition forms between normal color-perception and that of G. M. exist as congenital anomalies, but as transitory conditions in optic-nerve diseases such forms occur.

If a violet-blind patient has an affection of the optic nerve, the visual and color disturbances run the same course as in persons with normal color-perception. A case of this sort (unilateral traumatic atrophy of the optic nerve becoming

stationary) has been previously reported by me. With the affected eye the patient called the two colors that he saw in the chromoscope "red" and "blue," and matched them with orange and blue yarns; with the healthy eye his color perception was exactly similar to that of G. M.'s eyes.

There remains the supposition that in the form of violet-blindness described, components of the color system are present other than those existing in persons with a normal color-sense, and that these components lie closer together than the components in persons with a normal color-sense; but that within the shortened spectrum of the violet-blind the same relations exist as in persons with a normal color-sense. Whether the number of fundamental perceptions is three or four, one must hold to the idea that the fundamental perceptions are to be sought in the same parts of the spectrum in all individuals, and that color disturbances arise only through diminution in intensity or complete absence of definite fixed fundamental colors.

The symptoms of acquired progressive color-disturbances can only be explained by supposing that in the person affected the fundamental perceptions change their location in the spectrum, and two unite to form one.

I have already mentioned my so-called chromoscope, a device in which a white strip is seen through a prism, while outside objects are cut off from view. This chromoscope has proved in my hands to be the most convenient instrument for determining quickly and accurately the color sense of a person. All who have more than two fundamental color-perceptions with this instrument see practically only four colors, which lie in the spectrum at λ 641, 583, 510, and 428; for G. M., who is violet-blind, the four colors seen lie at λ 623, 583, 516, and 451. From this it appears that the four colors seen consist of two pairs of complementary colors; for me λ 641 and 510, λ 583 and 428 are complementary, and for G. M., λ 623 and 516, λ 583 and 451 are complementary. For me, red and marine blue, yellow and violet; for G. M., reddish orange and greenish yellow, yellow and indigo. The complementary color of yellow λ 583 is, for me, violet λ 418; for G. M., indigo λ 451. Thus the complementary colors

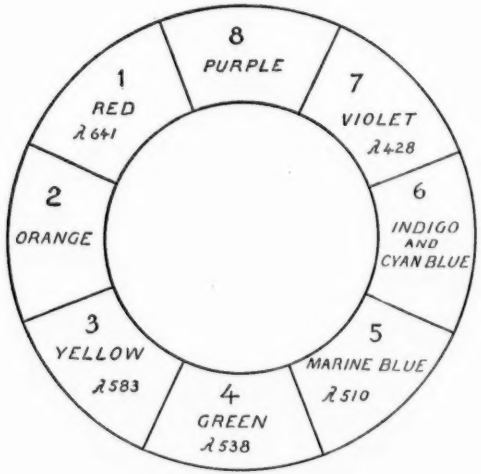

FIG. 3.

A color circle composed of the chromoscope colors as I see them, and the combinations of them in pairs as they lie near each other in the spectrum.

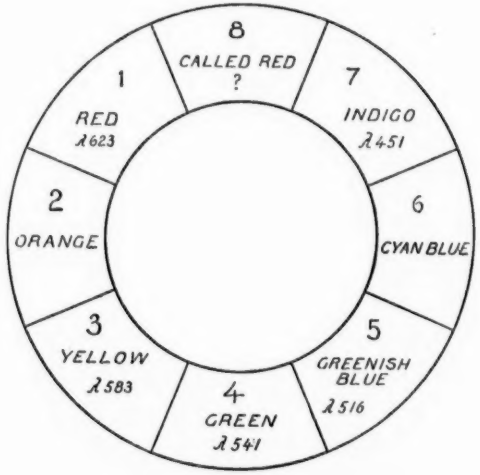

FIG. 4.

Color circle composed of the chromoscope colors as G. M. sees them, and their combinations.

are not always alike for all persons who see more than two colors.

Since the colors seen in the chromoscope are complementary in pairs, they possess the chief quality demanded in a system of four fundamental colors. They are, furthermore, evenly distributed through the spectrum. From their combinations all the colors of the spectrum can be produced; and they have the advantage over the colors of Hering's system, that they all are found in the spectrum. They are the colors 1, 3, 5, and 7 in the spectrum, and when arranged in a color circle it is found that the mixture of any two contiguous colors will produce the intervening color which separates the two in the normal spectrum, while the mixture of 1 and 7, red and violet, gives purple (Fig. 3). If a color circle is made up of the colors that G. M. sees in the chromoscope, the mixture of the contiguous colors would produce a series nearly corresponding to Hering's fundamental colors (Fig. 4); and for this reason I have suggested the possibility of Hering's visual system being related to that of G. M. G. M.'s red (1) and indigo (7), when mixed, produce to my eye a reddish purple. G. M. calls this simply red, but says it is not so yellowish as the red seen in the chromoscope.

It is easy to demonstrate that the colors seen in the chromoscope are, for the person using it, complementary. Those persons seeing two colors only, see blue and yellow or blue and reddish yellow, which, for them, must be complementary since they see no other colors.

The four colors that spontaneously present themselves in the chromoscope have all the characteristics demanded of fundamental colors, and I know of no fact or observation opposing the view that the colors seen in the chromoscope may be the fundamental colors for the person using it. Considered as fundamental colors, these differ from those of Helmholtz in being four instead of three, and from those of Hering in that they all are found in the spectrum, while Hering's red = purple. They differ from both the other series in not being absolutely fixed, but differing in different individuals, and, in cases of optic-nerve disease, differing at different times in the same individual. It is therefore

incomprehensible to me why my view has recently been described as an unsuccessful attempt to rehabilitate the Hering theory. Even if one will not accept the chromoscope colors as the fundamental colors, they are still of importance in judging of the color sense of the macula. If they are not fundamental colors, they are at least colors which are characteristic for the various color anomalies.

A word as to the relation of violet-blindness and ordinary color-blindness to normal color-perception. We have seen that both of these color anomalies may be found in persons who have previously had good color-perception, in case atrophy of the optic nerve develops. In such cases one can directly observe and follow a pressing together of the four chromoscopic colors until only one cold and one warm color remain, and finally these are lost, and no color is seen in the chromoscope.

If we assume that between those with normal color vision and those with congenital defects without pathological changes there is an intimate connection, there are two views to choose from. Either the color-blind have degenerated from a condition of normal color-vision, or the normal color-vision develops gradually through a stage in which but two colors are seen, thus reversing the process found in optic-nerve atrophy, and the color-blind are such as have remained in an imperfect stage of development. The latter view is in my opinion the correct one.

A form of pathological blue-blindness has been described by Koenig (*Sitzungsber. d. Berliner Akad. d. Wissensch.*, 1897). This is found in patients with chorio-retinitis and detachment of the retina and was spoken of in my book on the relations of the eye and its diseases to other diseases of the body and its organs (Wiesbaden, 1893).

The chief points of this paper are as follows:

1. Besides the well-known anomaly in which only two colors are seen there is another still more frequent anomaly — violet-blindness — in which the remainder of the spectrum is seen correctly, and at least three fundamental color-perceptions must exist.

2. This color anomaly accords neither with the Helmholtz nor with the Hering theory.

3. The chromoscope is a very convenient instrument for determining the color sense of the macula.

4. I myself believe that the four colors seen in the chromoscope are the fundamental colors for that particular individual. These may differ at different times in the same person if there is disease of the optic nerve.

5. According to their number and location in the spectrum I differentiate the congenital color-anomalies with normal anatomical and ophthalmoscopic conditions into: (*a*) two forms, each with two fundamental colors, the most fitting designations for which are: yellow-blue seeing and orange-blue seeing (= red-blindness and green-blindness of Helmholtz, protanopes and deuteranopes of v. Kries, and red-green blindness of Hering); (*b*) a form with as many fundamental colors as the normal-sighted possess, which is best designated as violet-blindness since this is its prominent characteristic, although there is a shortening of the long-waved end of the spectrum which, measured in wave-lengths, is almost as great as the shortening of the short-waved end (= perception of four colors with contraction of both ends of the spectrum).

6. All three of these forms of congenital color-anomaly are to be regarded as states of a less advanced development of the color preception. The latter develops in extra-uterine life in a way contrary to that in which it is lost in disease of the optic nerve. The three congenital anomalies of color are the three principal stages in the development of the color sense. Intermediate stages may at times come under observation, but these are very rare.

ON THE COLOR DISTURBANCE PRODUCED BY SANTONIN IN NORMAL AND COLOR-BLIND EYES.

By Prof. M. KNIES, Freiburg-in-Baden.

Translated by Dr. Ward A. Holden.

G. M., the violet-blind patient described in the previous paper, expressed himself as being willing that experiments with santonin should be tried on him, and this led me to repeat my experiments upon myself and also to undertake some on animals. We both found that the light sense did not suffer at all from the action of santonin either in the initial stage of violet-vision, which with G. M. was blue, or in the later period of yellow-vision. Therefore it was, according to v. Kries's discovery, highly improbable that the visual purple played any rôle in santonin poisoning, since, during adaptation in the dark, vision is essentially a function of the rods. Insufficient reproduction of visual purple would have lengthened the time of adaptation and increased the threshold, but neither of these changes was found.

Herein is a very marked difference between the yellow vision of santonin poisoning and vision through a yellow glass, which in many features are similar. In the latter case the time of adaptation in the dark is markedly lengthened and the excitation threshold is greatly increased. (The same is true for the visual and color disturbances in choroiditis and in retinitis of the outer layers and also retinal detachment, the condition being called "torpor retinæ".) The difference is clearly evident; the yellow glass cuts off the violet rays which most markedly act on the visual purple,

and therefore a greater quantity of non-violet light is required to exert a noticeable action of the light. In the santonin eye, however, even in experiments in the dark, exactly as much light falls into the eye as into other eyes, and if the visual purple is normally present the light acts on it in the same way as in a normal eye.

I also examined the retinas of a dog and a number of frogs after the administration of santonin. A small dog received 1.00 of potassium santoninate, after which it was kept for three hours in the dark and then killed.

The retinas when removed showed an intense purple coloring. This was also the case with frogs after the introduction of a crystal of about 0.2 beneath the skin of the back. Santonin poisoning, therefore, is without influence upon the visual purple and the function of the rods of the retina.

On ourselves we began with a preliminary experiment. I took 0.2 and G. M. 0.15 of potas. santoninate. In my case violet-seeing came on after forty minutes, and an hour later a perceptible yellow-seeing appeared, while G. M. noticed nothing. Light sense and adaptation were quite undisturbed, as they were in the following experiment.

Some days later each of us took 0.5. After an hour I saw violet whenever I suddenly regarded white objects or pale reflexes on a dark ground. The spectrum for me was at this time considerably lengthened at both ends, at the red end from λ 742 to 764 and at the violet end from λ 404 to 398.

An hour later yellow-seeing began, and the spectrum rapidly became shortened at both ends — at the red end to λ 721 and at the violet end to λ 411. In both stages the light sense was examined and found normal.

With G. M. the colored-seeing began fifteen minutes later, and was described as blue-seeing. The spectrum at its short-waved end was increased from λ 421 to 415, but I cannot say with certainty upon what this depended. Later his spectrum became shortened to λ 707 at the red end and to λ 423 at the other, although the shortening due to the santonin was much less marked than in my eyes.

The action of santonin on the visual organ is essentially a peripheric action limited to the cones of the retina, which, after a hyperæsthesia for violet and extreme red light, later becomes anæsthetic. Analogously, I found that the entire cutaneous surface of my body, after experiencing a feeling of warmth, became numb and remained so for a considerable time.

In the stage of yellow-seeing there is hyperæsthesia for the portion of the spectrum which is still visible. The yellow-seeing is not marked in a moderately light room, but is very evident whenever white objects are looked at. The central or cortical perception of color seems to remain undisturbed, for even in the stage of yellow-seeing shadows have the complementary violet color.

It is worthy of note that in santonin poisoning not only do both ends of the spectrum suffer—a fact which has long been known—but the shortening at the red end is even more excessive than at the violet. Nevertheless the color disturbance—first excitation, then anæsthesia—is much more pronounced for violet, because of the unequal distribution of the colors in the spectrum. The same is true in congenital violet-blindness, and in optic-nerve atrophy in which the perception of violet is the first to be lost. Santonin poisoning and optic-nerve atrophy are alike in causing little disturbance of the light sense, the latter, however, being much reduced when one looks through a yellow glass.

In seeing through a yellow glass no violet light falls on the normal cones (yellow vision) and rods (diminished light sense, because little, if any, of the visual purple is decomposed). In santonin poisoning lights of every sort fall upon the altered cones and normally functionating rods (normal light sense). In affections of the outer layers of the retina, such as chorio-retinitis, detachment of the retina, and the like, lights of every sort fall upon altered cones and altered rods, the former causing the disturbances of color perception and acuteness of vision, the latter the disturbance of the light sense known as torpor retinæ.

All this holds good for those with normal color-perception and for the pure congenital color anomalies. For myself

and for the violet-blind G. M. the symptoms of santonin poisoning are entirely analogous. I have not had the opportunity of experimenting on those who perceive only two colors. But in such persons there would be first a lengthening of both ends of the spectrum with blue-vision, followed by a longer-lasting condition of yellow-vision, with which both ends of the spectrum are shortened.

From what has been said it is easy to explain the puzzling cases of blue-vision as a minor degree of the more frequent yellow-vision in persons who are violet-blind.

TRANSFIXION OF THE IRIS.[1]

By Dr. HUGO ASCHHEIM.

Translated by Dr. Ward A. Holden.

AT the Heidelberg Congress of 1896, Fuchs read a paper on "Transfixion of the Iris." Now a series of cases have been operated upon and we wish to describe again the indications for the operation and results.

The operation is designed to take the place of iridectomy in cases of *iris bombé* after exclusion of the pupil, and, in the words of Fuchs, is done as follows: "An incision is made through the cornea 1–2 *mm* from its outer margin with a Graefe knife that is not narrow, the handle being so held that the blade lies parallel to the surface of the iris. The knife is then passed through the prominent iris, and a counter-puncture is made near the nasal margin of the cornea. The knife is then pushed in until the entire width of the blade occupies the counter-puncture section, when it is withdrawn."

The reports of cases which follow are arranged in two groups. In the first are cases in which the eye had been operated on previously for cataract, in the second group are the remaining cases. In Case 5, transfixion was done on both eyes. Besides these thirteen operations, five others were done on out-patients, and of these an accurate history cannot now be obtained.

I.—FIVE CASES OF TRANSFIXION OF THE IRIS IN APHAKIC EYES.

Case 1.—L. W., aged sixty. October 31, 1891.

The patient had a senile cataract in the left eye, which was

[1] From Prof. Fuchs's clinic in Vienna.

extracted with iridectomy April 23, 1891. When discharged his vision was $\frac{5}{18}$, but later, in consequence of thickening of the capsule, it decreased to $\frac{5}{36}$.

October 6, 1891, a discission was made, and three days afterwards the tension became increased and the iris was pushed forward. October 10th, an iridectomy downward was done. At this time the patient could count fingers at 2.5 *m*. Since the temporal half of the iris again bulged forward and tension increased, on October 31, 1891, transfixion of the prominent portion of the iris was done, and the anterior chamber was restored at once.

November 10th the patient was discharged with a normal anterior chamber and V = $\frac{5}{36}$; in the temporal segment of the iris was a V-shaped cleft.

Case 2.—J. S., aged fifty-one. January 27, 1896.

In January, 1894, a senile cataract was extracted from the left eye, and in December of the same year discission was done. For two weeks the patient has complained of pricking in the eye operated upon.

On examination the iris was found to be pushed forward. It was atrophic, and it exhibited a small coloboma upward, whose outer margin was not distinctly visible. The tension was increased. With + 12. + 2. c, V = $\frac{5}{8}$. Transfixion was done January 28th, after which the iris returned to its place and the tension became normal. Below the pupil there could be seen a perforation in the iris 3 *mm* long.

Case 3.—Schl., aged thirty-eight. March, 1896.

The patient was first seen in December, 1892, having at that time a soft cataract in the left eye. This was twice discinded in the following year, and later a puncture was made. The patient then had V = $\frac{5}{6}$ with + 12. D. Six months later a delicate membrane was discinded, and in January, 1896, a thickened capsule was split. At that time V = $\frac{5}{6}$ with + 11. D.

In March, 1896, the patient suddenly noticed severe pain in the operated eye while on a journey. When he returned it was found that the iris had been forced forward against the cornea, and that the pupillary margin was adherent to the capsule of the lens, the pupil being free from opaque membrane. T + 3.

March 4th transfixion was done, and the anterior chamber was restored. In the inner half of the iris was a perforation as large as a pin head, and three days after the operation the patient's vision

was again $\frac{6}{9}$ with + 11. D. This condition has remained to the present time.

CASE 4.—Antonie St., aged seventy-five. November 4, 1897.

On January 13, 1897, a senile cataract was extracted from the left eye without iridectomy. Some days later the iris prolapsed into the wound, and a portion was excised. A slow iritis which followed caused a blocking of the pupil and the coloboma, and necessitated an iridectomy, which was done in the horizontal meridian with a Graefe knife, leaving a black pupil which was almost circular.

But since the iris at its margin was adherent to the capsule, it became forced forward and the tension increased, so that on May 14th transfixion was done, with permanent result. Now, almost a year later, the perforations are clear, the anterior chamber is deep, and the tension is normal. With + 10. + 2. c 10° N, V = $\frac{5}{18}$.

CASE 5.—Therese H., aged forty-five. June 8, 1896.

In February, 1895, the patient was first seen with a sympathetic iritis in the right eye, and in March, 1896, an iridectomy was done downward. The new pupil closed again, and when the patient returned in June the cornea was dull, the anterior chamber shallow, the iris like blotting-paper, the coloboma closed by a membrane, the pupil, in which the cloudy lens was visible, surrounded by a grayish line where the iris was adherent to the capsule, and blocked by a membrane, T + 2, V = movements of fingers at 30 *cm.*

On June 9th, extraction according to Wenzel was done, but a free pupil was not obtained, the iris again pressing forward. Therefore, on June 22d, transfixion was undertaken, and the chamber again assumed a fair depth. On June 30th an iridectomy was done, and at first a fine black coloboma was obtained. Later this closed, the iris again bulged forward, tension became increased, and the eye was lost.

II.—EIGHT CASES OF TRANSFIXION OF THE IRIS IN NON-APHAKIC EYES.

CASE 6.—Marie B., aged fifty-seven. Jan. 10, 1897.

In January, 1896, an inflammation of the right eye was ushered in with redness and pain, and passed off after a month, leaving the vision cloudy. Twelve days before her first visit the patient noticed severe pain in the eye at night, and on examination there were found conjunctival and ciliary injection of the ball and

haziness of the cornea, with numerous precipitations on the posterior surface of the cornea. The anterior chamber was shallow in places owing to localized projections of the iris, which had lost its markings, and had undergone a grayish green discoloration. The pupillary margin was everywhere adherent to the anterior capsule, and the pupil, as far as one could discover through the turbid aqueous, was blocked by a membrane. T + 1. V= movements of the hand before the eye.

On January 10th transfixion was done, after which the anterior chamber was restored. A small perforation could be seen near the pupil. The tension was slightly below normal. Later two perforations could be seen in the external segment of the iris, tension was normal, and the patient was discharged, January 20th, with the ability to count fingers near the eye.

Ten days later the patient returned with a fresh attack of iritis which passed off under treatment, and on February 16th an iridectomy was done on account of the pupillary membrane. On February 18th the patient could count fingers at 1 *m.*

Case 7.—Francisca Sw., aged forty-two. February 1, 1897.

About five months before, an inflammation of the right eye appeared after an injury. Since then the patient has noticed a gradual decrease in vision, accompanied by severe pain in the right half of the head and the perception of colored rings on looking at a flame. The present condition is as follows: marked conjunctival and ciliary injection, and the cornea unevenly curved, dull, and with an opacity external to its centre, corresponding to an adhesion of the pupillary margin to the cornea. The iris is discolored and without lustre, and irregularly pushed forward. T + 1.

Transfixion was done on February 3d, resulting in two perforations in the upper segment of the iris, and the bulging was permanently relieved. On February 10th there was still a slight increase in tension.

Case 8.—Anna G., aged fifty-six. February 13, 1897.

In the autumn of 1893 the patient was under treatment for a left irido-cyclitis and received iodide of potassium and atropine which she used whenever the eye became red and painful. In the past week, the pain growing worse, the patient returned. The anterior chamber was found to be of varying depth, owing to an irregular pushing forward of the iris. The latter is discolored and dull. From the minor circle radiating yellowish stripes run toward the

periphery; in these regions the iris is less prominent. The pupillary margin is adherent to the anterior capsule, there is a pupillary membrane, and the tension is + 2.

On February 16th transfixion was made, after which the anterior chamber was restored and the tension lowered. In the upper portion of the iris two perforations could be seen, one triangular and one round.

CASE 9.—Carl H., aged thirty-three. November, 1897.

The patient had had trouble with his eyes for three weeks and was received with a slow irido-cyclitis on both sides which had led to adhesion of almost the entire pupillary margin of the iris. On November 23d an iridectomy upward was made, with good result. Three days later the anterior chamber was found to be full of blood and a brownish-green discoloration of the cornea took place from the presence of blood-coloring matter, as was first accurately described by Treacher Collins. Gradually the cornea cleared from the periphery and it was seen that the iris was being forced forward against the cornea. At the same time the tension became increased.

In this case transfixion was done on January 10, 1898, the knife being entered near the inner margin of the cornea and passed through the inner portion of the iris, but not pushed to a point of counter-puncture, because the opacity of the cornea prevented its control. Nevertheless the result was excellent, since the chamber became deep and remained so, and tension returned to the normal. The small perforation in the nasal portion of the iris remained permanently open.

CASE 10.—Albertine P., aged twenty-three. February 6, 1898.

In November of the preceding year the left eye became painful and teared. On examination the ball was found to show conjunctival and ciliary injection, the cornea and adjacent portion of the sclera were prominent, and the surface of the cornea was stippled. The anterior chamber was partly obliterated by localized protrusion of the iris. The latter was atrophic and discolored, and united by the pupillary margin to the anterior capsule. In the pupil was a fine membrane. T + 2. Perception of light.

Immediately after transfixion the anterior chamber was restored, and the increase in tension disappeared.

Some days later, however, while the chamber remained and the perforations were open, the iris and lens became pushed forward, and the tension again increased, so that iridectomy was done, February 10th.

CASE 11.—Franz H., aged twenty-eight. November 4, 1897.

Two years before, the patient had an alternating inflammation in the two eyes, which later passed off. Two months before, an inflammation appeared in the right eye, and a week before in the left. When examined, the right cornea was dull, the anterior chamber of normal depth, the iris dull, the pupil displaced upward, unresponsive, and blocked by a membrane; the left cornea was dull, the iris pushed forward irregularly, and the pupillary margin adherent to the capsule. R V = fingers at 30 *cm*; L, at 20 *cm*.

The day after the patient was admitted, a localized protrusion was found in the right iris, and transfixion was therefore made in both eyes. The following day the anterior chamber in each eye was deep, and one perforation could be seen in the right eye, and four in the left.

In the right eye the transfixion was of temporary benefit only, since in the next two weeks the perforation became closed as a result of a fresh iritis, the iris again bulged forward, and tension became increased. On November 17th, an iridectomy upward was done with good result; but the coloboma and pupil were later drawn toward the upper margin of the cornea by iritic exudations, and on January 18th a second optical iridectomy had to be made inward.

In the left eye the result was permanent; the four perforations remained open, the chamber remained deep, and the tension continued normal. On January 18th, the pupil having become blocked by a membrane, an iridectomy was done. When discharged, the patient's vision was R $\frac{3}{6}$, L $\frac{6}{5}$.

CASE 12.—Marie W., aged twenty-six. February, 1895.

The patient has for three years had trouble with her right eye, which now is ectatic and blind.

A week before an inflammation appeared, and now there are signs of severe irido-cyclitis. The cornea is dull, the aqueous turbid, the anterior chamber partly filled with a gelatinous exudation; the iris is hyperæmic and dull, and there is a fresh exudation in the pupil; the tension is increased, and V = fingers at 20 *cm*.

Later the iris bulged forward, and transfixion was done. The immediate effect was good, and the chamber was restored, but the iritis continuing, the perforations closed, and on March 16th it was necessary to do an iridectomy. Some time after, a second iridec-

tomy was necessary, and then an iridotomy, but all were without permanent result, and the eye gradually became atrophic.

The transfixion of the iris was done here in eighteen cases. In all there was a bulging forward of the iris, due to exclusion of the pupil, followed by increased tension; in some of the cases the eye was aphakic. The purpose of transfixion is to restore communication between the anterior and posterior chambers, return the iris to its normal position, and reduce tension. It matters not whether one or many perforations are made, provided any one of them remains permanently open.

Perforating wounds of the iris do not close if no iritis is set up. This observation led Fuchs to try transfixion, with the results here recorded. In three of these cases the result was not permanent, because irido-cyclitis again broke out. In certain conditions transfixion seems preferable to iridectomy. My thanks are due to Prof. Fuchs for suggesting this subject and assisting me in the preparation of the paper.

THE EXAMINATION OF TWO CASES OF OLD SPECIFIC CHORIO-RETINITIS.[1]

By Dr. G. NAGEL.

Translated by Dr. Ward A. Holden.

(*With three figures on Plates XVIII.-XIX. of Vol. XXXVI., German Edition.*)

THE number of microscopical examinations of eyes with syphilitic disease is not large. Besides some short reports by J. Hutchinson, Bader, Klebs, Fürstner, and Pagenstecher, I have found the following accounts in the literature. Edmunds and Brailey[2] make the following summary statement as to syphilis of the interior of the eye: The choroidal vessels seem not to be altered, while those of the retina, both in acquired and congenital syphilis, are thickened, probably from inflammatory processes. The central artery of the retina does not appear to become affected so long as the syphilis is not complicated with another disease. In syphilis, as in glaucoma, a varicose condition of the capillaries is often found, and in cases of chorio-retinitis the retinal vessels are often pigmented.

Nettleship,[3] in a careful study of the pathological changes in syphilitic choroiditis and retinitis, comes to the general conclusion that in syphilis neither does the retinitis depend exclusively upon a choroiditis (Förster), nor does the contrary relationship exclusively hold good (Ole Bull), but rather both views are probably equally correct. As regards particular histological changes, Nettleship found accumula-

[1] From the University Eye Clinic at Breslau.
[2] *Royal London Ophth. Hosp. Reports*, 1882, vol. x.
[3] *Ibid.*, 1887, vol. xi.

tions of cells in the choroid and between the choroid and the retina, and a thinning of the choroid after absorption of the inflammatory products, and in many cases an increase in the pigment epithelium. These changes begin constantly in the choroid and never upon it. "So far as I have seen, the choroidal changes always begin in, never upon, this tunic, the exudations upon its retinal surface occurring later from extension inward of luxuriant cell growth."

In the retina there is a general thickening of all layers, brought about by a round-celled infiltration which has not the disposition, observed in the choroid, to occur in nodules. The thickening of the nerve-fibre layer was especially well marked, and many nuclei were found in the optic nerve. In the arteries and small vessels of the retina the walls were thickened and pervaded with nuclei. In the final stage of a severe and extensive case the retina was thinned, some of its layers could not be distinguished, and the blood-vessels all were ensheathed in pigment and the lumen often closed. "Such a retinitis pigmentosa in consequence of choroiditis represents a condition which is often found in eyes blinded by chronic inflammation not of syphilitic origin."

Uhthoff,[1] in a syphilitic woman of forty-seven, found extensive changes in the iris, but in the choroid only slight changes in the form of partial atrophy. The pigment epithelium was well preserved throughout. In the retina the smaller arteries and veins had their lumen more or less occluded by a proliferation of small cells.

Bach[2] found inflammatory changes in the adventitia and intima of the arteries and capillaries, the veins remaining unchanged. The vessels of the choroid were free from inflammatory changes, but there were patches of diffuse inflammatory processes in the supporting tissue.

From this, Bach concludes that syphilitic retinitis is independent of choroiditis.

Rochon-Duvigneaud,[3] in a child of two years dead of hereditary syphilis, found in the macula of the right eye an indrawn scar in which choroid and retina were firmly united.

[1] *Graefe's Archiv*, xxxix., 1, 1893.
[2] *Arch. f. Augenheilk.*, xxviii., 1894.
[3] *Arch. d'opht.*, xv., 1895.

About this scar was a slight detachment of the retina, beneath which was an albuminous liquid containing degenerated elements of the retina, round cells, and pigment. In the choroid there were foci of infiltration in the chorio-capillaris. The author believed that the primary lesion was in the choroid in the region of the macula where the choroidal capillary network is particularly rich and retinal vessels fail.

Dor[1] reported on both eyes of an infant which died in the course of labor. Many hemorrhages were found in the retinas, and also pigmented patches. The syphilitic nature of this case was questionable, and it is well known that retinal hemorrhages are very frequent in new-born infants that are otherwise healthy.

For the two cases which I now wish to report I am indebted to Prof. Uhthoff.

CASE R. — **Clinical picture of syphilitic chorio-retinitis that had run its course. Unilateral; ophthalmoscopically similar to retinitis pigmentosa: the vessels in the periphery surrounded by pigment; no actual foci of choroidal atrophy, but numerous small foci of pigmentation in the periphery of the retina, some stellate, some circular. Death from typical paralysis. Pathological examination revealed localized adhesions between retina and choroid. Sclerosis and pigmentation of the retinal arteries, as in retinitis pigmentosa. More pronounced pathological changes in the retina than in the choroid. Vitreous practically unaffected.**

The examination of a large number of microscopical preparations was made. In the anterior segment of the ball there was nothing abnormal. In the posterior segment the sclera was normal. There were post-mortem detachment of the choroid from the sclera, and of the retina from the choroid. The detachment of the retina was not general, however, and there were many adhesions between the two. At such points the pigment epithelium was mostly wanting, and it was absent also from the most posterior portions of the retina, and also from the detached portions; but since the pigment epithelium is so readily detached, its absence from the portions of the retina artificially detached is not significant.

The choroid seemed normal wherever the overlying retina was detached, but about the points of adhesion it was in high degree degenerated, the chorio-capillaris being entirely destroyed.

[1] *Arch. d'opht.*, 1896.

The retina presented more advanced changes than the choroid. At many points the outer layers were wholly obliterated. Chiefly in the inner layers, and particularly on a level with the blood-vessels, were regularly formed masses of irregularly arranged pigment. At the points of adhesion between retina and choroid one sometimes gained the impression that the pigment of the pigment epithelium had wandered directly into the retina (Fig. 1, Pl. xvii.). In some preparations the pigment that was frequently found near or in the vessel walls suggested the ophthalmoscopic bone-corpuscle arrangement seen in retinitis pigmentosa.

Further than this, there were thickening of Muller's fibres, and marked changes in the vessels. The vessels near the posterior pole appeared normal, but those elsewhere had thickened walls, often due to a diffuse inflammatory infiltration of the entire thickness of the wall, and at some points having led to complete transformation of the vessel into a connective-tissue cord. Pigment was found in the walls of many vessels in all layers, but it varied in amount and distribution. Often the vessels had a complete sheath of pigment, a condition which with the narrowing of the lumen of the vessel is that found in retinitis pigmentosa.

The pigment found in the retina has everywhere the well-known character of that from the pigment epithelium, consisting of small rod-shaped and crystalline particles.

CASE **G., an idiot fifteen years of age, hereditary syphilis, both upper and both lower extremities paralyzed and contracted, patient unable to stand. Amaurosis, discs not atrophic. Picture of complete atrophy of the choroid with marked pigmentation of the entire fundus. On autopsy extensive changes were found in the cerebro-spinal system due to syphilis.**

Microscopic examination: Marked alterations in the choroid with complete disappearance of the chorio-capillaris, while the retina with its vessels was unchanged. (Fig. 3.)

Sections of the posterior segment of the ball were examined microscopically. In the optic nerve and sclera nothing pathological was found, but in all the sections alterations in the pigment epithelium were manifest. At places the layer was wanting, in others greatly thickened and sending processes out into the choroid. Only a few small accumulations of pigment were found in the retina, and these only in the outer layers. The large masses of pigment in the choroid lay at points where there were infiltrations and thickening of the retina, and retina and choroid were adherent in such manner that the outer layers of the retina

had disappeared, as well as the chorio-capillaris of the choroid, while the layers of large vessels in the choroid were mostly well preserved, and the retinal vessels were normal.

CONCLUDING REMARKS.

As regards the relation of syphilitic choroiditis and retinitis, most authors agree with Förster that the retinitis is dependent upon the choroiditis. Nettleship believes that this holds good for a portion of the cases, but for other cases he believes that the retinitis is the primary affection, as Ole Bull believes. Interesting in this regard is the report of Duvigneaud, whose case of syphilitic chorio-retinitis was, without doubt, primarily one of choroiditis.

Our second case also is essentially one of choroiditis, while in our first case the retinal changes were much more extensive than the choroidal. It is thus possible that cases differ, and that the choroidal and retinal affections do not always stand in the same relation to each other.

In order to take up a further point in the pathology of this form of disease, we have spoken in detail of the changes in the vessels that were found in our cases. In general, taking into consideration these two cases, and the others already reported, it appears that the changes are inflammatory in the widest sense of the word, with a marked tendency to sclerosis.

Michel and Alexander would explain all the syphilitic affections of the interior of the eye as being due to specific endarteritis. By endarteritis specifica we understand a pathological condition in which, together with proliferation of the endothelium, a peculiar infiltration of the vessel walls takes place, the whole picture differing from the ordinary inflammatory alterations of vessels. That changes in the vessels may usher in syphilitic chorio-retinitis is unquestionable, but whether this at first has the character of specific arteritis is not yet proven. In our cases the changes were too old to throw any light upon this matter.

Finally, I should like to say a few words in regard to the similarity of old specific chorio-retinitis with retinitis pigmentosa as it appeared in our Case R., both clinically and microscopically.

Wagenmann [1] concluded on the ground of experimental investigations that in retinitis pigmentosa the choroid was the primary seat of the trouble; a conclusion which was supported by Bürstenbinder,[2] who had opportunity to examine microscopically a case of retinitis pigmentosa in its incipiency. He found inflammatory changes in the choroid.

When we recall that Wagenmann found destruction of the chorio-capillaris with consecutive involvement of the outer layers of the retina, the similarity of these changes with those found in our Case R. is at once apparent. Wagenmann stated expressly, in opposition to Wedl and Bock [3] who believed the pigment to be choroidal, that the pigment was derived from the pigment epithelium.

In our case also we saw that the pigment had, without any question, wandered into the retina from this layer, leaving gaps at various points. The pigment granules, furthermore, had the form characteristic of retinal pigment.

There exists then much anatomical similarity between old specific chorio-retinitis and retinitis pigmentosa; but, on the other hand, the specific process in our preparations is characterized by the peculiarity that the changes in the choroid and retina do not correspond in the measure in which this is the case in retinitis pigmentosa.

1. At many points there are pronounced focal changes in the choroid, above which the retina is but slightly affected.

2. At many points the retina is considerably thickened without a corresponding abnormality on the part of the choroid.

This agrees with the clinical experience that in many cases which at first seem to resemble retinitis pigmentosa, a more careful ophthalmoscopic examination reveals focal changes in the choroid, and a diagnosis of old chorio-retinitis must be made. The microscopic examination furnishes a finer test, and probably those cases of specific chorio-retinitis which exhibit no focal changes in the choroid and resemble retinitis pigmentosa would, when examined microscopically, appear very different from the typical retinitis pigmentosa.

[1] *Graefe's Archiv*, xxxvii., 1.
[2] *Ibid.*, xli., 4.
[3] *Path. Anat. des Auges*, 1886.

Furthermore, periarteritic changes as described by Uhthoff and Bach are not found in retinitis pigmentosa, while in specific cases they furnish the proof that the vascular changes may be of manifold character, just as they are elsewhere in the body.

If one wished to be entirely accurate in the matter of nomenclature the designation retinitis pigmentosa should be applied not as at present to the simple pigmentary degeneration of the retina but rather to the chorio-retinitis, and particularly the syphilitic form that leads to pigmentation. It has long been recognized that typical pigmentary degeneration is not an inflammatory process.

The study of fresh cases of syphilitic inflammation of the eye would be of value in determining accurately the nature of the early vascular changes.

My thanks are due to Professor Uhthoff and Professor Axenfeld for their kindness in furnishing me the material and aiding me with their advice.

A CONTRIBUTION TO THE THERAPY AND PROPHYLAXIS OF EXPULSIVE HEMORRHAGE DURING CATARACT EXTRACTION.[1]

By Dr. CARLO PEIRONE, Turin.

Slightly Abridged Translation by Dr. Ward A. Holden.

THE first to describe hemorrhage threatening the existence of the eye as a complication of cataract extraction seems to have been Wenzel in 1779. Beer also had seen such cases, and he writes that he always operated on them before witnesses, although he does not tell us what signs led him to expect such complications beforehand. In the present paper it is not my intention to give an historical résumé of the various cases reported, but, taking as a text two cases seen in Dr. Peschel's clinic, to consider the method by which in the future one may be able to combat or even prevent this complication either in both eyes or at least in the second eye when the first has been lost from hemorrhage.

From the cases reported, we learn that the hemorrhage may occur immediately after the scleral or corneal section (Thomas and Thompson), in the later course of the operation, immediately after the operation while the dressings are being prepared, some hours later (Warlomont, Meyer, Knapp, Simi, Badal, Gasparrini), or even a day or more after the operation (Girard, Panas, Da Gama Pinto). In cases of the last category the anterior chamber had not closed, or the wound had reopened, allowing the escape of aqueous and thus lowering the tension, or if the wound remained closed the hemorrhage was slight and recovery took place as in

[1] From Dr. Max Peschel's clinic at Frankfort-on-the-Main.

Becker's case and in Rydel's atypical case (1897), in which on the fifth day the anterior chamber became filled with blood which soon absorbed and recovery was complete. Such hemorrhages in the anterior chamber during the period of recovery after extraction may come from the iridectomy. In the first few days after the excision of the iris the new circulation does not take place through capillaries but through large anastomoses between arteries and veins, from which hemorrhage may readily occur. An immediate cause is the displacement of the iris as the anterior chamber is restored, or the dilatation of the pupil from atropine. The hemorrhage does not come from the canal of Schlemm since, as Becker has shown, this is not involved in a proper section of the cornea.

The hemorrhage may recur at regular intervals for several days (Girard), or it may recur after having begun and ceased in the course of the operation (Becker).

The hemorrhage may be accompanied or immediately followed by obstinate vomiting, epileptiform attacks (Berry), a feeling of heat in the orbit, severe pain in the eye, and flashes of light. One objective symptom is constant, namely, loss of vitreous. An intra-ocular hemorrhage may follow loss of vitreous, as it were, *ex vacuo*, but in our case the sequence was the opposite, since the hemorrhage was the active force that caused the vitreous to escape.

As to the different methods employed, Da Gama Pinto, Simi, Becker, Girard, and Warlomont observed hemorrhages after a small flap downward had been made, Sedan after a flap upward, Badal after a modified Graefe's extraction, Gasparrini after extraction with iridectomy, Hilemann after simple extraction, and Warlomont and others in spite of preliminary iridectomy, while Da Gama Pinto and Girard obtained favorable results with the last method.

Where is the location of the hemorrhage? Warlomont and Van Duyse found in an enucleated eye, besides complete loss of vitreous and detachment of the retina, detachment of the choroid from hemorrhage into the supra-choroidal space, and they therefore regard the choroid as the source of the hemorrhage. Becker saw blood pouring

through the pupil and hence concluded that it must come from the posterior chamber. In another case he observed after the expulsion of the lens a prolapse of vitreous without rupture of the hyaloid membrane and without hemorrhage, but blood could be seen in the depth of the vitreous. The eye was hard and glaucomatous and the patient suffered from severe pain and saw phosphenes. Atrophy of the ball followed without suppuration of the cornea. Proudfoot (1889) found on examining an enucleated eye that the source of the hemorrhage was a branch of the central artery of the retina which was dilated and torn at a point near the disc. Fage observed a large coagulum in the anterior segment of the ball, which separated the sclera from the ciliary body and had its origin in the choroid. Goldzieher concluded that the hemorrhage arose from the long posterior ciliary arteries, since these are the only vessels which in their intra-ocular course lie entirely behind the choroid, and since the choroid is always found intact in the enucleated eyes. Dufour also believed the hemorrhage to be supra-choroidal. Fromaget and Cabannes (1895) found the choroid and ciliary body pervaded with blood, and they believed the source to be the large vessels of the choroid, confirming their ideas by the examination of a later case (1896). Panas in one case found a great coagulum between the sclera and choroid. The fact that in the membranes forced out by the hemorrhage there are found at times bits of the retina only and not of the choroid does not disprove the view that the hemorrhage arises in the latter. In such cases the blood broke through the choroid and collected between the choroid and retina.

The examination of the vessels of the eye revealed, according to Girard and Mooren, a fatty degeneration of the walls and atheroma. Da Gama Pinto believed that the walls had been friable. Becker in one case found a telangiectasis involving the entire corresponding half of the face, and supposed that in the interior of the eye were similar varicosities which ruptured when the tension of the ball decreased during the operation. Van Duyse experimented on rabbits and found that, in spite of ligation of the jugular

vein and section of the sympathetic, no hemorrhages occurred when the vessels were normal although the blood pressure was thus greatly increased. He believed the causes of hemorrhage to be an increased tension of the vessels and arterio-sclerosis which might be primary or consecutive to the long-continued high tension. Fromaget and Cabannes found the larger vessels and the capillaries in part widely dilated but without alterations of their walls. Panas describes the retinal vessels in his case as being thickened and almost obliterated by desquamation of the intima, and the vessel walls in the choroid much thickened. Rahmer and Jacques introduced a new hypothesis. The ciliary arteries are intimately connected with the sclera where they pass through it, so that after being ruptured they cannot retract, but must remain open like the vessels of the diploë. This hypothesis is strengthened by the fact that enucleation stops the flow of blood at once since the vessel walls can then collapse.

Apart from the local conditions found in the retinal vessels, the examination of the vascular system in general will give one information as to the state of the retinal vessels. Da Gama Pinto states that in his patient simple compression of the skin on the back of the right hand was sufficient to cause ecchymoses. Hilemann found in his case that a fresh choroiditis had preceded, weakening the vessel walls, and that the patient suffered from acute rheumatism of the joints. Simi found the radial artery greatly thickened and transformed into a nodular cord, and the patient cachectic; in another case there was abdominal venous stasis. Van Duyse speaks of atheroma of the vessels or hæmophilia. He believes also that the vasomotor innervation has some influence and therefore calls these hemorrhages "essential." Terson, Panas, and others seek an analogy in atheroma, in which the general blood-pressure is increased and the heart becomes hypertrophied. Graefe observed in these patients a loss of elasticity in the skin so that folds in the skin remained for a long time.

Certain eye diseases predispose to hemorrhage. Thus, one of Da Gama Pinto's patients was highly myopic. But

glaucoma is frequently the underlying cause of hemorrhage. Spalding found iridodonesis as the result of choroiditis in his case. In Risley's patient, aged eighty-two, he found hypermature cataract, iridodonesis, and synchysis of the vitreous. In Knapp's case there was a complicated Morgagnian cataract.

The consequences of the hemorrhage were usually panophthalmitis and loss of the eye without suppuration of the cornea. Fienzal, Proudfoot, Fage, and others enucleated the eye in the course of the panophthalmitis. In many cases the eye was lost from atrophy without destruction of the cornea; such were reported by Knapp, Becker, Steinheim, Bowman, Warlomont, Graefe, etc. Girard was forced to enucleate one eye for sympathetic ophthalmia in the other. And a few observers (Becker, Girard, Gasparrini) obtained a relative recovery with greatly diminished vision and contracted field. Knapp in one case had such a favorable outcome, but two weeks later fresh hemorrhages occurred in the anterior chamber and vitreous, and finally both eyes were lost from iritis and phthisis bulbi.

As to prophylaxis and therapy, Dufour advised the injection of morphine with apomorphia. The narcotic and nauseating effects cause the hemorrhage to cease, and these drugs can be used also prophylactically. One case so treated retained a slight amount of vision. Da Gama Pinto, Power, Derby, and Warlomont advise against chloroform or ether since some of their cases seem to have been brought about in this way. Jackson advised in suspicious cases that the patient be operated on in a sitting position with the feet hanging out of bed. Preparatory iridectomy is to be employed generally in glaucomatous eyes; in the non-glaucomatous it is not always a protection against hemorrhage. Spalding operated thus with good result on the second eye after the loss of the first. Da Gama Pinto saw the anterior chamber fill with blood during the preliminary iridectomy. The extraction was successfully done later but there was some hemorrhage into the vitreous.

Abadie advised continued compression of the carotid during and for twenty-four hours after the operation. The

pressure bandage is commended by some. Terson in cases of arterio-sclerosis prescribes iodide of potassium for some time before the operation and for three or four days before uses veratrum viride to decrease the heart's action and lower the blood pressure. Chloral is used at night. Warlomont, after losing both eyes from hemorrhage, concluded that needling only should be done in the second eye when the first had been lost from hemorrhage. Fage and others recommend ergotin injections into the temples and complete rest. Liebreich refuses operation of the second eye, preferring the relative blindness of the cataract patient to complete amaurosis.

I wish now to report two cases operated on by Dr. Max Peschel.

Madame R., aged seventy-five, came to Dr. Peschel in February, 1895, with a ripe cataract in the right eye and advanced cataract in the left. There was general atheroma of the arteries of the body and some hypertrophy of the left ventricle. Over the aorta was heard a rough systolic murmur. Traces of albumen in the urine. For some years attacks of giddiness. The eyes proved to be normal functionally, and in spite of the danger of complications an extraction was decided upon. Some days later the right eye was operated on by the modified linear extraction of von Graefe. The eye was washed out with bichloride solution 1:5000 the night before and a moist dressing was applied which was left undisturbed until the following day. After being again disinfected, cocaine and eserine were instilled and a smooth operation with iridectomy was done. There was slight hemorrhage from the iris. Scarcely had the lens been removed when the patient complained of severe pain in the eye, the margins of the section gaped, and normal vitreous protruded with a red reflex behind it. Immediately a clot of blood appeared in the wound. The eye was at once covered with a tight sublimate dressing and Dover's powder was given. As is usual in these cases the dressing was soaked with blood and had to be renewed in the evening. The inner membranes of the eye were protruding from the wound as an irregular blood-stained mass.

The treatment consisted in attempting to prevent infection by applying a moist bichloride dressing. This was changed twice a day and was always stained with blood. On the third day the

prolapsed mass was cut off close to the ball with scissors. The two lips of the wound remained separated by a solid, bloody, pigmented mass. The dressing still showed traces of blood a month later. Since the eye was kept antiseptic, purulent panophthalmitis was prevented, but the ball atrophied until, two months later, only a stump was left with a deep retraction corresponding to the corneal section.

The prolapsed matter that was excised was examined partly fresh, partly after being hardened in Müller's fluid. It consisted of a portion of the ciliary body and iris with the adjacent choroid and retina. One could distinguish a portion of the periphery of the retina about 3 *mm* long. In sections the retina was seen to be separated from the choroid by a blood clot. In the choroid and in the ciliary body were parenchymatous hemorrhages partly diffuse, partly localized, forcing the normal elements apart. The retina contained blood, its elements were altered, and there were many small vacuoles indicating œdema. All the tissues showed an infiltration with leucocytes, densest near the vessels. The choroidal arteries were atheromatous, their walls thickened, the intima and media in a state of granular fatty degeneration or calcification, the adventitia sclerosed, the lumen often narrowed. Single venous trunk also showed calcareous degeneration. The capillaries were dilated and overfilled with blood. All these alterations were less marked in the vessels of the retina. A small arterial branch in the choroid was found to be obliterated.

In this case the hemorrhage was due to rupture of the atheromatous vessels, aided by the compensatory hypertrophy of the heart, the determining cause being the sudden lowering of tension at the operation. The length of the corneal section is of importance, for prolapse of vitreous and rupture of the hyaloid membrane occur much less readily when the section is small.

When the cataract in this patient's other eye was ripe, a different method of operation was selected. With a discission needle an N-shaped incision was made deep in the lens. After seven days a small lance was passed obliquely through the cornea, so that the wound would gap as little as possible, and the masses of swollen lens substance were removed by pressure and with the spoon. The zonula and posterior capsule remained intact. On the evening of the same day

the anterior chamber was restored. There was no iritis. After a month the atropine was discontinued, and after two months vision, with the correcting glass, was $\frac{10}{10}$.

A second, analogous case was that of a cook, aged seventy-two, who came to the clinic the 1st of March, 1896, with a ripe cataract in each eye. There was general emaciation and atheroma of the arteries, with marked hypertrophy of the left ventricle. The patient suffered habitually with dyspnœa, palpitation, dizziness, and pain about the heart. The condition of the eyes was absolutely normal, except for the cataracts.

On March 10th an operation with iridectomy was attempted on the left eye. When the capsule was divided, the patient noticed a severe pain in the eye, with phosphenes. Then the lens escaped and a bead of vitreous prolapsed. In the vitreous could be seen a bloody tumor, covered with the inner membranes of the eye. These were cut through with de Wecker's scissors, allowing the blood to escape and the membranes to collapse.

The hemorrhage continued for six days, and later iritis led to an exudation blocking the pupil. Two months after the operation the tension was normal, perception of light existed, and projection was wanting only from the upper-inner portion of the retina which had been detached.

A month after this operation the problem arose as to the operation on the other eye. The capsule of the lens was divided by N-shaped incisions. An iritis developed, with a slight increase in tension. A small vertical section of the cornea was made with a lance obliquely and the liquid portion of the lens evacuated. The following day the eye was quiet and the pupil dilated well with scopolamine. Five days later the section was opened with a spatula and the bulk of the lens was evacuated. A few cortical masses were left, the eye remained somewhat sensitive, and for two weeks a small hyphæma persisted. Two months after the operation, with the correcting glass vision was $\frac{10}{10}$. The vitreous was clear, and the fundus was normal, except for patches of senile choroiditis about the disc.

The method of operation described led thus in two cases to very satisfactory results, and I would recommend it for the first eye when hemorrhage is feared, and for the second

eye when the first has been lost from hemorrhage. Discission of a ripe cataract is much less to be feared than discission of an immature cataract. The method of operation is exactly that used for the removal of the transparent lens in high myopia.

These two case histories show that expulsive hemorrhage is not always due to glaucoma. And it is in these cases only that the operation is indicated. In glaucoma, it goes without saying that a preliminary iridectomy should be done. In support of our view we may cite those cases in which, even after preliminary iridectomy, the eye was lost from hemorrhage (Gasparrini, Warlomont, Van Duyse), a palpable proof that the operator should seek for a safer method.

The bibliography of the subject may be found in Spalding's paper, these ARCHIVES, xxv., 1, p. 92 (1896), there being wanting only references to the paper by Terson (1894) and Willot (1891).

REPORT OF THE TRANSACTIONS OF THE SECTION OF OPHTHALMOLOGY AND OTOLOGY AT THE NEW YORK ACADEMY OF MEDICINE, MAY 21, 1900.

By Dr. J. H. CLAIBORNE, Secy.

The evening was devoted to presentation of specimens and cases, and discussion upon the **cause, prevention, and treatment of prolapse of the iris following simple extraction of cataract.**

Dr. Arnold Knapp described a **method of preserving macroscopic specimens of eyes** based on Kaiserling's procedure (*Virchow's Archiv*, 1897, vol. cxlvii., p. 389) of preserving the natural colors of pathological specimens. The method is the following:

1. The usual fixation and hardening in 4% formol for 4–5 days.
2. Bisection of eyeball.
3. The eye is placed in a solution of 80% alcohol for 6–12 hours until the color of the blood is restored, and then in the following mixture:
4. Acetate of potash, 200.
   Glycerine, 400.
   Water, 2000.

In which the eye may remain an indefinite time, until ready to be preserved in the same mixture in a Priestley Smith jar. By this method the natural color of the eye, the color of the blood, the transparency of the cornea, and to some extent of the lens, are permanently preserved. The formol preservation of eyes has many advantages over the old jelly method, but the detrimental action of formol on the color of the ocular structures is well known.

Two specimens, one of a **gumma of the ciliary body,** the other of **multiple cysts in a detached retina,** preserved after this method, were then exhibited.

Dr. M. L. FOSTER exhibited a boy of six, in which there was **exophthalmos and paralysis of the external rectus.** Kroenlein's operation was done. Around the optic nerve was found a pear-shaped tumor whose apex pointed toward the ball. This was evidently cystic in character, and had a bluish, thin wall. The tumor was aspirated and cerebro-spinal fluid was withdrawn. The sac could not be completely removed, but portions of it were taken off. The optic nerve was clearly demonstrated. The subsequent result was good, although internal strabismus existed after the operation. At the time of presentation there was optic atrophy. In the beginning, however, there was no optic neuritis. The exophthalmos disappeared. Dr. Pooley noticed that there was anæsthesia of the cornea, which he attributed to injury to the ciliary nerves.

Dr. SKEEL presented a case of **corneal injury** due to a broken china cup. There was prolapse of the iris, which was excised. A flap from the conjunctiva below was drawn up to the top of the cornea, covering the wound. On the second day the anterior chamber was restored. On the fifth day the conjunctiva was œdematous, and subsequently the flap became adherent to the cornea. It then contracted somewhat, and the patient at time of presentation had vision of $\frac{2}{3}\frac{0}{0}$. Dr. Skeel proposed to remove later the conjunctival flap covering the cornea.

Dr. CHAMBERS presented a case in which there was **absence of the auditory canal on either side,** with rudimentary or imperfectly formed auricles. As to family history, the father's brother had a deaf-and-dumb child.

Dr. W. A. HOLDEN presented a **glass composed of red, green, and blue,** which was constructed for the purpose of viewing the eclipse of the sun which was shortly to occur. Dr. Holden stated that this combination of colors diminished the luminosity of the sun, but permitted the normal color to remain. Dr. Skeel remarked that it might be a good thing in the belt of total eclipse, but at this position, New York, the corona could not be seen. Dr. Holden replied that he had constructed it for some friends who were going to Norfolk to view the eclipse.

Dr. EMIL GRUENING opened the discussion on the **cause, prevention, and treatment of prolapse of the iris** in these cases, and said that the cause of the prolapse of the iris was undoubtedly pressure on the eyeball during the operation, pressure of the bandage after the operation, increased intravascular pressure, and,

altogether, all conditions which tend to increase the intraocular pressure. In order to prevent prolapse of the iris, it was the custom of some to discard the speculum during the operation. Others resorted to hypodermatic injections of morphine, in order to quiet the general nervous system of the patient. Some thought that the section of the cornea, if perpendicular to the surface, predisposes to prolapse. He said that the elasticity of the iris played an important rôle, and was also to be considered. There was no doubt that some irides are more elastic than others, and have a tendency to return to their first position when the cause of the extrusion is removed. In order to obviate the conditions favoring protrusion of the iris, a number of devices have been adopted, which might be divided into the following classes :

1st. Reduction of intraocular tension, for example, by cocaine, pilocarpine, eserine.

2d. Strengthening of the weak point—that is, the point of least resistance. To this end, he referred to Kalt's suture, the conjunctival bridge, and von Milligen's contact glasses.

3d. Diminution of the area of impact by incising the iris after the manner of Schweigger, or buttonholing the iris, after the manner of Chandler (Boston, Mass., Char. Eye and Ear Inf.), Flueger, or Bel-Taylor.

4th. Lowering the general intravascular pressure and, secondarily, intraocular pressure. In his own experience, he had found that the lowering of the intravascular pressure during and before the operation by the instillation of a 4% solution of cocaine, and after the operation by the instillation of a 10% solution of cocaine, had acted most happily, by inducing an hypotony of the globe, and subsequently a retention of the iris. The use of cocaine, he maintained, in the way he had mentioned, does not cause opacity of the cornea, nor dilatation of the pupil subsequent to the operation, as has been stated.

The treatment of prolapse of the iris, he said, might be discussed under the following heads :

1st. Conservative treatment.

2d. Treatment by cauterization (actual cautery).

3d. Reduction of the iris.

4th. Abscision of the iris.

5th. Excision of the iris.

Of all these methods, it was the fifth, excision of the iris, which he was accustomed to practise, not only in prolapses which oc-

curred within twenty-four hours after the operation, but also in those that occurred at a later period. By excision he wished to be understood a cutting out of the iris from between the angles of the wound, so that after the completion of the operation there would remain a clean iridectomy in the shape of a key-hole pupil. It has been asserted by some that the frequency with which there is prolapse of iris following the lower section of the cornea is due to the fact that the iris falls by virtue of its own weight, namely, by gravity. On one occasion, when Schweigger was operating for cataract, and was making an inferior or lower section, a bystander made this remark. Schweigger replied that, in his opinion, the iris did not prolapse by virtue of its own weight, but on account of the influences from the rear, or from other causes. This notion had impressed Dr. Gruening, and he used this as an introduction to his remarks.

The discussion was continued by Dr. C. S. BULL, who said that Dr. Gruening had covered the ground so well that he could only speak of the methods of preventing the prolapse of iris, and the treatment of it when it occurred. He said there was no one rule for all cases. It was necessary for the surgeon to study each case, and so get at the prophylaxis for each one. He thought it proper to always study the action of the iris. He was accustomed to do this repeatedly under the use of a 5 % solution of cocaine, and in this manner decided whether a simple extraction or combined extraction should be employed. If the iris was rigid under the use of cocaine, he thought it likely to be rigid after the operation, and so was likely to be prolapsed. Under these circumstances, he was not accustomed to do a simple extraction. If the patient was an unruly person or easily excited, he was accustomed to do an iridectomy, and if there was plus tension after the use of cocaine he also did an iridectomy. It was his habit to use cocaine after the manner stated by Dr. Gruening. He examined the eyes twenty-four hours after operation. If he found a prolapse, partial or complete, he would first try and replace it by a spatula. If not possible to do that, he would excise it. If a prolapse occurred after twenty-four hours, he preferred to leave it unmolested. He thought this rule was a good one, because prolapsed irides often flattened. To sum up his treatment, he said if prolapse occurred after twenty-four hours he excised. If after three days, he left it alone.

Dr. KNAPP said he divided the causes of prolapse of iris into five classes:

1st. Any rupture of the suspensory ligament before or during the operation.

2d. Prolapse of vitreous, either immediately after or subsequent to the operation.

3d. If the corneal section fell beyond the limbus.

4th. Injury during the first days after the operation.

5th. Restlessness of any kind during and after the operation.

The prevention he also divided into five classes :

1st. Iridectomy before or after the expulsion of the lens in defective zonulæ.

2d. The greatest caution was necessary in opening the capsule, lest the zonula be ruptured, especially in hypermature cataracts, in which dislocation of the lens and capsule, or rotation of the lens inside the capsule may occur.

3d. Insufficiency of corneal section or of capsular opening. The capsular opening, especially in hypermature cataract, should be sufficiently peripheric, otherwise the edge adjacent to the section will be kept in a pouch at the periphery, and there will be a breech delivery by turning of the lens, which is laborious but not bad.

4th. Watching of the patient during the first days and nights by an extra nurse in restless patients.

5th. Masks of different kinds.

As to treatment, he was careful during the operation to cleanse the pupillary area of remnants, reduce the iris (if it did not resume its position by its own elasticity) by gentle rubbing, or with a blunt-pointed probe. If he could not obtain a central, round pupil, he made a small iridectomy and reduced the columns of the coloboma carefully.

He inspected the eye a day after the operation. If there was prolapse, never so small, he seized it with forceps, abscised it, and reduced the limbs of the coloboma. He had tried reduction of small prolapses, but as they mostly reappeared, he had given up reducing them. When the prolapses had occurred longer than one, or, at the highest, two days before they were noticed, he did not interfere until the wound had completely healed, when he abscised them like any other partial staphyloma.

Dr. T. R. Pooley agreed with Dr. Gruening in the opinion that a rigid or non-resilient iris is especially apt to be the cause of prolapse. He thought the discriminations made by Dr. Bull, in a careful examination of the behavior of the iris under cocaine, of value, as were also those of Dr. Knapp, in reference to the

character of the cataract. He would add to the category which Dr. Knapp had given, such cases of cataract as were not yet completely mature, as unfavorable for simple extraction because of the liability of producing prolapse of the iris in the attempts made to extrude the soft lens remnants. He could not see that the instillation of a ten per cent. solution of cocaine would prevent prolapse, but, on the contrary, would rather favor it.

In order to prevent prolapse of the iris, not only was it important not to make the wound too small, but also not to place it near the periphery but well within the sclero-corneal margin. The most frequent cause of prolapse after extraction he thought to be due to disquietude on the part of the patient during the first night after operation, by preventing closure of the wound, and in many other cases even trauma inflicted by the patient's hands.

In regard to treatment, he expressed surprise that so little had been said about the treatment of prolapse during operation, and said that the efforts to replace the iris which made necessary the frequent introduction of instruments in the wound and into the anterior chamber, were to be deprecated; unless the iris replaced itself or was readily replaced, iridectomy should be done. In case of the appearance of prolapse after extraction, he would excise the iris where it occurred after the first twenty-four hours, but in cases occurring later, leave it to nature. One case was cited of prolapse after the third day which healed without a cystoid scar and the visual result was very good.

In conclusion, reference was made to the danger and difficulty of excising the iris in unruly patients through the loss of vitreous, hemorrhage, etc. He gave the history of one case in which the loss of vitreous made an imperfect iridectomy and the result was a bad one. He also mentioned a case of a colleague's in which, through the unmanageability of the patient, vitreous was lost, irido-dialysis caused, and hemorrhage took place, the eye being lost. The use of an anæsthetic was therefore urged as the safest and wisest procedure when attempting to excise the iris in cases in which prolapse took place after operation.

Dr. Richard Derby said that he hardly felt he had a place in this discussion, and he was afraid his remarks would be considered far afield. It was his custom always to perform the combined operation, that is, the operation with iridectomy, and therefore he did not feel that he could properly discuss the "cause, prevention, and treatment of prolapse of the iris following simple extrac-

tion." It was in order to prevent the accidents and contingencies that had been so clearly outlined by the gentlemen who had already spoken, that he had adopted the combined extraction, and while he felt that he did not follow the mode of the day, he nevertheless was in the line of safety.

Dr. J. S. PROUT said he believed that the use of the bandage after extraction does injury by interfering with the coaptation of the lips of the corneal wound, and is, therefore, a factor in the cause of prolapse of the iris after simple extraction. To avoid this unequal pressure Michel advocated closing the lips of the wound by a light plaster and no other dressing, not being afraid to leave the eyes so little protected. He referred to the suggestion of Dr. H. W. Skerry, of Brooklyn, who was accustomed to use a wire mask in addition to the plaster dressing. This mask is figured and described in the *American Journal of Ophthalmology*, January, 1891. He found, however, that the plaster interfered with the application of liquids to the eye, and so on March 11, 1890, in the case of a man of eighty, with chronic conjunctivitis, instead of using the plaster he inserted a silk suture through the edges of the lids after the cataract extraction. In this way he was able to instill a weak solution of bichloride of mercury between the lids. He found this method very good. The suture was removed in four days. A single aseptic silk suture will do the work. He thought it well to insert the suture before the operation, as there might be some injury done from sudden resistance or starting on the part of the patient after the operation. In *La Clinique Ophtalmologique*, April 10, 1900, Dr. Jocqs reports having used fine silver wire for this purpose with good result.

Dr. DAVID WEBSTER divided the causes of prolapse into two classes, non-traumatic and traumatic. The first class he also described as the mechanical method. He said that regenerated aqueous humor filled the space formerly occupied by the lens and also the anterior chamber. The tension then becomes increased. The aqueous runs out and the iris falls out with the aqueous. Patients are then accustomed to feel pain and the sensation of a gush of water. When this history is told him, he is accustomed to take off the bandage, and he usually finds prolapse of the iris. 2d, traumatic causes are due to the rubbing of the eye by the patient, or some other form of injury. He saw no way of preventing traumatic prolapse of the iris except by using a mask. Immediately after the operation he was accustomed to use both a

bandage and a mask over the bandage. When the anterior chamber had filled completely, he used simply a wad of cotton and a mask over it. Under any circumstances, he was accustomed to find about 4 % of prolapses of the iris. He had replaced the iris with a spatula, and had seen good success, but he had abandoned it. On several occasions he had destroyed the prolapse with the actual cautery, but he was not satisfied with that method, as severe iritis had followed. To excise or not to excise, is the question. If much inflammation attended prolapse, he thought it unwise to excise. He waits until the inflammation disappears, and then, if the prolapse is not flattened, he cuts it off, but he cuts off only that part which is outside. Twenty-four hours after the operation he is accustomed to do an iridectomy.

Dr. SUTPHEN said he was accustomed to follow the habit of Dr. Bull, viz., observing the pupil in a good light. In consequence of this method, he had come not to fear prolapse as much as he had before. He allows the patient to sit up on the day following the operation, and used a cloth shield over the cotton dressing.

Dr. CALLAN said he thought prolapses were mostly due to traumatism, and that cases in which there was a deep anterior chamber were less likely to prolapse than those in which there was a shallow anterior chamber. He thought that Dr. Webster was right: The cause of the prolapse lay in the leaking and outflow of the aqueous humor. The position of the head was also a matter of importance. In shallow eyes he considered there was more chance of prolapse, because the resistance to the outflow of the aqueous humor was greater. He also considered that violence to the sphincter of the iris was a cause; likewise when the cataract was large. More force would have to be employed to deliver it, and in such cases the iris was likely to be prolapsed. If, after the extraction, the pupil remained oval, it was likely that there would be a prolapse. He always tried to replace the iris, whether 24, 36, or 48 hours after the operation,—it made little difference. He thought the suggestion of Dr. Pooley, that anæsthesia should be employed when a second operation was necessary, was a good one. In unruly patients he used cotton and a mask.

REPORT ON THE SECTION OF OPHTHALMOLOGY, BRITISH MEDICAL ASSOCIATION.

By C. DEVEREUX MARSHALL, F.R.C.S.

IPSWICH MEETING, AUGUST 1 AND 2, 1900.

DR. W. A. BRAILEY, PRESIDENT, IN THE CHAIR.

Presidential Address.

Dr. W. A. BRAILEY, ophthalmic surgeon to Guy's Hospital, took as the subject of his address Ocular Headaches—that is, headaches in association with refractive and muscular errors. While the great majority of headaches were, he said, independent of the eyes, it was a matter of general acceptance that ocular errors produced headaches, though by no means in all cases. Were there any errors especially effective in the causation of headaches or in producing any particular form of headache? Muscular errors were by far the most important, though, of course, these must be ultimately of nervous origin. Ocular movements were intrinsic (pupillary and accommodative) and extrinsic (of the recti and obliqui muscles). The pupillary movements were unimportant except as slightly influencing glare. The accommodative movements were bound up with the great majority of ocular headaches.

It was a general law that the larger the ocular error the less the effect produced on the head, the reason being that a great defect of accommodative power led to its abandonment, the patient seeing as best he could without it. So, also, uncorrected presbyopia was rare as a cause of headache, except just at its commencement. It might cause strain and burning but not headache. A highly hypermetropic patient would read close with poor vision but no aching. So also patients with high hypermetropic astigmatism. Similarly, great inequality of refraction gave comparatively

little trouble, the worse eye being abandoned to disuse and muscular action being regulated by the better one. High degrees of myopia and myopic astigmatism produce little effect, distant objects hardly being seen, while near vision could be remedied largely by adjusting the distance. But low degrees of hypermetropia and hypermetropic astigmatism were often causes, especially when they led to an excess of effort both in amount and duration beyond that which was needed, as with weak muscles or hypersensitive nervous supply, this spasm of accommodation continuing in distant vision, and even under retinoscopy in the dark. But both spasm and headache were more produced by moderate inequality of refraction, especially if astigmatic and most of all by astigmatism with asymmetry of the axes. The ciliary muscle appeared to act unequally on the two sides in correcting this or a muscle even in different parts of its circuit in remedying astigmatism or asymmetry of axes. Evidence of this was seen when atropine revealed astigmatism on one side or both, increased its amount, or altered the axis of the correcting lens in amount less than 90°. So astigmatism, often unequal-sided, might become manifest when presbyopia began, and refractive defects revealed themselves to retinoscopic examination in eyes blind with fundus changes, such as optic atrophy, in proportion beyond that observed in average-seeing eyes. The above refractive errors caused aching in the eyes, often passing presently or on the following morning into the brows, immediate aching in the brows, temples, the back of the head, and occasionally also headache of the type of migraine. Treatment by appropriate glasses was of extreme value. Errors of the extrinsic muscles produced headache, but less than of the accommodative muscles, though more migraine, more giddiness, and more general distress. Here, also, the rule held that the larger the amount of error the less the disturbance, the explanation being that in considerable degrees of strabismus the image fell on the peripheral and so less acutely seeing retina.

But another potent factor in headaches was the tendency to binocular vision, and so we had another rule—the stronger the tendency to binocular vision the more headache produced by an error of the recti and obliqui muscles. Binocular vision varied much in its strength in different subjects. Possibly there were natural differences related to the centres as much as to the muscles, for he had seen such indisposition run through families.

But binocular vision appeared to be generally made rather than born. Babies often squinted irregularly till after a few weeks of life they got their yellow spots gradually to accord. He had met cases where, instead of bringing them into harmony, the child had developed another retinal point to work with the opposite yellow spot in an eye congenitally squinting, though with fair concomitant movement. Rectification of such apparent squint by tenotomy would produce diplopia. Other cases were common where the two eyes remained quite independent; for example, where there was congenital want of power of both external recti from central defect, diplopia was absent and the child saw to his left with the right eye, and *vice versa*. Binocular vision was not strongly established by the early age at which ordinary concomitant squint arose; so diplopia, though present, gave no trouble, and, indeed, was little noticed even in the comparatively rare cases where each eye had normal refraction and vision. But to take the case of a paralytic strabismus—for example, of the superior oblique—suddenly arising in an adult: here was much disturbance, and the same with cases where, when the two eyes had been used for different purposes—that is, one for near and the other for far,—they suddenly attempted by lenses and prisms to unite the images, the results being distress, giddiness, migraine, and other headache. But extrinsic muscles might, like the ciliary, have large latent defects, and he suspected that many obscure cases of headache had their solutions here.

Besides individual tendencies other influences altered the disturbing effect of ocular errors: (1) age, the errors being most potent between the ages of ten and forty-five years; (2) sex, women being the greater sufferers; and (3) nationality. His experience did not extend much beyond the English people and the citizens of the United States, the latter of whom were eminently affected by these errors, so that a series of medical men and operators had arisen to take charge of that special sub-section of practice. Doubtless there were other influences, such as occupation and temporary states of the nervous system.—*Brit. Med. Jour.*

Discussion on the treatment of **chronic glaucoma.**

This discussion was opened by Mr. RICHARDSON CROSS, who based his opinion on the results obtained in forty-seven cases of the disease, which had come under his care in private practice. He had frequently found difficulty in distinguishing some from cases of optic atrophy, and the perimeter was of special value in

these cases, the contraction on the nasal side being very characteristic. There was no doubt as to the fact that progressive glaucoma would cause blindness sooner or later through atrophy if left to itself, and in the majority of cases drugs were incapable of saving the sight alone.

He looked upon iridectomy as the most useful operation, and in a simple chronic case it was comparatively easy to do, and very much easier than in acute cases. In some exceptional cases, in which the presence of the disease was doubtful, it could sometimes be cured by drugs alone, and he mentioned a case which, after treatment in this manner, showed no increase of tension after prolonged dilatation with cocaine. The treatment of chronic glaucoma by drugs alone in nearly all cases, however, led only to the gradual deterioration of vision.

He then showed numerous charts of fields of vision which had been taken both before and after operation, and treatment with myotics, and in many of the cases the fields of vision had improved after the disease had been arrested. In very early cases he thought that sclerotomy done with a keratome, as recommended by Snellen, was very useful, and he preferred a keratome to a Graefe knife, as he thought that a more peripheral wound could be made. He was strongly in favor of early operation, and he did not think that anything but harm could come in the majority of cases if drugs without operation were relied on.

Mr. JOHN HERN thought that the one thing needful was an early diagnosis, and the sooner an iridectomy was done the better the chance of a good result. He thought that there was a close relation between optic atrophy and glaucoma. His practice was to do an iridectomy as soon as possible, and to follow this up by establishing a communication between the anterior chamber and the vitreous by passing backwards through the coloboma above the lens a narrow double-edged knife, and then to slightly rotate this in order to make the opening a little larger.

Mr. WORK DODD was by no means inclined to look so favorably upon iridectomy as a cure as Mr. Cross appeared to do, and this led him to remove the superior cervical ganglion of the sympathetic. He described minutely his case, which immediately responded by the pupil becoming contracted, and the tension diminished. He thought that in a suitable case this operation was justifiable.

Dr. CARL GROSSMANN said that he preferred pilocarpine to

eserine. Massage had given good results, especially in the form of a mallet driven by a galvano-motor. One mode of treatment which was especially useful when a patient was averse to the more serious operation of iridectomy was the subconjunctival injection of sterilized salt solution, which had given very encouraging results in his hands.

Mr. BOWER fully agreed that the sooner an operation was done the better, but thought that occasionally a case might be kept in a practically stationary state by the use of myotics, and he mentioned a case he had had under observation which had not got materially worse during six years. In very early cases with good vision he thought that the operation of sclerotomy, as suggested by Mr. Cross, might be very valuable, as the immediate result on vision would not be so marked as if a piece of the iris were to be removed.

Mr. G. A. BERRY had seen many early cases kept in check for years with pilocarpine, but he was strongly of opinion that in cases which had entered the confirmed stage the sooner iridectomy was done the better. He had seen cases in which, ten or fifteen years after the operation, the vision had undergone no deterioration. He thought that the worse prognosis was in those in which there existed a central or paracentral scotoma. He preferred iridectomy to sclerotomy, and did both with a keratome. The best results were obtained in those cases which healed with a flat scar. Scleral puncture he had tried, as recommended by Priestley Smith, as a preliminary to iridectomy. He had never excised the superior cervical ganglion of the sympathetic.

Mr. DEVEREUX MARSHALL thought that considerable damage might be done by the operation recommended by Mr. Hern, both to the lens and also to the ciliary body.

The PRESIDENT thought there was no reason to believe that the disease would progress, unless the tension were relieved. He thought that there were many fallacies in the perimeter. While coarse tests with a rapidly moving object, whether the finger or a 10 *mm* square, show a good field, yet fine tests show very great contraction of the field, and this would explain easily the considerable enlargement of the field that may undoubtedly occur under myotics. With regard to prognosis, although he did not consider it good, yet in cases on which he had operated early he had never had a bad result. When, however, the fields failed and the disc atrophied, he thought that, as a rule, the disease progressed in spite of all treatment.

Mr. SYDNEY STEPHENSON read a paper on the **etiology of phlyctenular eye affections** and, according to his experience, 20 to 25 per cent. of the eye patients at a children's hospital suffer from this disease.

There is, nevertheless, no general agreement as to the etiology of the cases. Mr. Stephenson had collected 669 cases, met with in hospital practice, and analyzed them, with special reference to three main points: (1) the frequency of an associated eczematous inflammation of the skin; (2) the existence of a tuberculous diathesis; (3) the influence of zymotic ailments in exciting phlyctenular disorders.

He found that of the total number of cases eczema was present, or had been present, or was known to have appeared later, in no less than 53.06 per cent. He was therefore inclined to regard phlyctenulæ as an ocular manifestation of eczematous inflammation.

The author also pointed out that eczema affected also the mucous membrane of the nose, lip, tongue, and palate. The oral lesions had hitherto escaped description. They commenced as vesicles, and speedily formed small circular or oval ulcers, which soon healed.

Mr. Stephenson found tuberculous tendency, or tubercle, in 31.98 per cent. of his 669 cases. He looked upon a marked tendency to phlyctenular disease as an indication that the patient was likely to become tuberculous — that is to say, if he did not already suffer from tubercle. In 13.45 per cent. of his cases measles, whooping-cough, chicken-pox, or scarlet-fever was assigned as the cause of the eye mischief. Mr. Stephenson regarded phlyctenular disease as due remotely to the tuberculous diathesis, and immediately to an eruption of eczema on the surface of the eyeball. The exciting cause might be of a general nature, as bad hygienic surroundings or measles, or of a local character, as slight injuries to the eyeball.

Mr. KENNETH SCOTT described a case of **dacryo-cystitis aggravans** which occurred in a woman, a native of Upper Egypt, who came to him with double dacryo-cystitis, which on the left side had attained an enormous size and resembled a malignant tumor of the face. He opened it and evacuated a large quantity of pus and mucus. The patient did well, and in five weeks she had quite recovered.

Mr. W. WATSON GRIFFIN related a case of **leuco-sarcoma** of

the iris which occurred in a young lady aged nineteen. In the left eye there was a growth, visible in part to the naked eye through the pupil, which pushed the iris forwards at the lower and outer side. The lens was displaced inwards and partially opaque.

Mr. Griffin diagnosed a malignant growth and excised the eye. On examination it proved to be a leuco-sarcoma with very marked hyaline changes. He thought that in a case treated so early the prognosis should be good. The points of chief interest were the youth of the patient, the extensive hyaline changes, and the dense white appearance.

The discussion on **lachrymal obstruction** was opened by Mr. G. A. BERRY, who referred to the great divergence of opinion held by surgeons as to the best method of treating this affection.

He was inclined to think that there was a great tendency to overtreat these cases, and this was due to the common belief that the cause of the trouble was in nearly all cases due to obstruction in the nasal duct, whereas in reality it was catarrh of the sac and duct. He condemned the use of large probes, as these frequently caused a stricture at the opening of the canaliculus into the sac, and this was especially likely to occur when the lower canaliculus had been slit. He preferred in nearly all cases to slit the upper one, as the direction of the channel thus made lay more in a line with the duct and there was consequently less stretching at the mouth of the sac and less laceration of the surrounding parts when the probe was passed. Owing to the fact that removal of the sac generally produces but little epiphora, it was obvious that faulty excretion was not the chief cause of the trouble, but probably hypersecretion of tears, due to an irritation which originated in the sac and possibly in the duct also.

In *early cases* Mr. Berry advocated slitting the upper canaliculus and then probing at intervals of a fortnight or longer, when usually three or four probings were sufficient. As a rule, syringing was unnecessary except in those cases in which the sac was overdistended, and then this, followed by pressure over the sac, was very useful.

Should an abscess develop, this was best treated by opening the sac by slitting one or both canaliculi, or, if necessary, opening it on the face.

In conclusion, Mr. Berry advised early interference in catarrhal conditions of the sac, viz., by slitting the upper canalicu-

lus into the sac and then probing at shorter intervals with a medium-sized probe. In dacryo-cystitis, free incision with good drainage was usually indicated ; this must be followed by opening of the canaliculi and in some cases by probing at fairly long intervals. In chronic cases, or those frequently recurring, complete removal of the sac was probably the best treatment. Large probes, frequent probings, and the use of styles were in his opinion to be avoided, while removal of the lachrymal gland or any portion of it was altogether uncalled for.

Dr. CARTWRIGHT said he avoided as far as possible the slitting of canaliculi, as it caused needless damage and impaired their function. In advanced cases, some could be cured by small probes, while for others gold styles were necessary. He thought that syringing was useful, but he had never seen any advantage arise from the use of hollow styles, nor had he ever seen a case in which it was necessary to remove the lachrymal gland.

Mr. KENNETH SCOTT, in cases of chronic inflammatory thickening, preferred the use of rather large probes and the syringe. He thought that lead styles were the best, and he had never seen harm arise from slitting the canaliculus.

Dr. ST. CLAIR THOMSON approached the subject from the standpoint of a rhinologist. In the cases he had examined he had not found any disease of the nose to account for the trouble, though he thought the primary infection of the duct and sac might come from the nose. He showed a drawing of a section of the nasal cavity to illustrate the large diameter of the lower end of the duct and also the large space between the inferior turbinate bone and the outer wall of the nose.

Mr. BOWER thought that overtreatment was responsible for much that was unsatisfactory. He never used styles and very seldom probes. In acute cases he considered the best treatment was to open the abscess and allow the case to quiet down, but if this failed he preferred to dissect out the sac.

Mr. ERNEST CLARKE thought that simple cases were best treated by dilating the canaliculus and syringing. He strongly condemned the use of large probes. In cases of stricture he recommended the slitting of the lower canaliculus and the removal of its ocular surface so as to accommodate a nail-headed gold style.

Mr. RICHARDSON CROSS saw no harm in slitting the canaliculus, and in severe cases he opened both. If a stricture existed, the

probe must be used, but he thought there was no advantage in using a large one.

Mr. SYDNEY STEPHENSON was convinced that in difficult cases there was nothing so good as large probes, and he preferred rapidly to dilate with Theobald's probes, the largest being 4 *mm* in diameter. Mucoceles in new-born children usually yield to pressure over the sac and evacuation of its contents.

Mr. MADDOX recommended the attachment of a simple nipple to the nozzle of a Weber's syringe for the injection of cocaine. When a style was indicated he advised the use of a soft one, such as the end of a fine silk catheter, which should be worn for a few days before placing a permanent metal one in the duct.

Mr. ENSOR, in severe cases, rapidly dilates under an anæsthetic with Couper's probes and then puts a lead style in.

Mr. WORK DODD drew attention to the importance of attending to the general health.

Remarks were also made by Mr. BLAIR and the PRESIDENT, Dr. BRAILEY.

Mr. BERRY, in reply, stated that he had never seen anything but harm arise from the use of large probes.

Mr. ERNEST CLARKE read notes of a case of **cerebral tumor with optic neuritis** in a man aged thirty-six. The right disc showed post-neuritic atrophy, while the left was a typical choked disc with 3 D of swelling but no hemorrhages. The presence of a tumor in the left frontal lobe was diagnosed. As the patient became rapidly worse, the skull was trephined by Mr. Arbuthnot Lane. The brain bulged greatly when the tension was relieved, but the patient died the next day. At the autopsy the tumor was found where it was expected, and it proved to be a **psammoglioma**.

Dr. FREELAND FERGUS read a paper on some forms of **optic-nerve disease, probably of sympathetic origin.** He related five cases in which injury to one eye was followed after a few weeks by contraction of the field of vision in the other. This contraction was always accompanied by a diminution in visual acuteness. In only one of the cases mentioned was there anything at all resembling an ordinary case of sympathetic ophthalmitis. He urged that in all cases of injury to one eye a close watch should be kept on the condition of the optic nerve of the other. This was to be done not merely with the ophthalmoscope but also by the constant examination of the field of vision and of

the minimum of light sense. Their two functions depend in part on the healthy condition of the optic nerve, and defects in them often showed lesions of the optic nerve before they could be detected with the ophthalmoscope.

The PRESIDENT asked if tension as a possible cause of failure of vision had been excluded. Remarks were also made by Mr. CROSS and Mr. BERRY. In reply, Dr. FERGUS said that he had carefully eliminated tension as a cause of failure. He thought that possibly the onset of an attack of sympathetic ophthalmia might be predicted by observations on the field of vision.

Mr. E. E. MADDOX demonstrated a stereoscope for the exercise of eyes after operations on the muscles, in which each eye could be shut off from seeing by means of small screens worked by electro-magnets inside the instrument. He also showed some new needles, with round holes for the suture, which could be threaded more readily than those of the ordinary pattern. For advancements he had had made some silk sutures, colored green and black.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FIRST QUARTER OF THE YEAR 1900.

By Dr. ST. BERNHEIMER, in Vienna; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin;

WITH THE ASSISTANCE OF

Dr. G. Abelsdorff, Berlin; Prof. E. Berger, Paris; Dr. Swan M. Burnett, Washington; Dr. Dalén, Stockholm; Dr. J. Herrnheiser, Prague; Prof. Hirschmann, Charcow; Dr. Krahnstöver, Rome; Mr. C. D. Marshall, London; Dr. P. von Mittelstädt, Metz; Prof. Da Gama Pinto, Lisbon; Dr. C. H. A. Westhoff, Amsterdam; and others.

Translated by Dr. Ward A. Holden.

Sections I.–III. Reviewed by Prof. HORSTMANN.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

1. Haab. Atlas and essentials of ophthalmoscopy and ophthalmoscopic diagnosis, with 149 colored and 7 black-and-white illustrations. Third enlarged edition. Lehmann's hand atlases, vol. vii. Munich, 1900: J. E. Lehmann.

2. Birnbacher. The pathological histology of the human eye. First portion, 5 plates. Leipsic, 1900: Veit & Co.

3. Wilbrand and Sänger. The neurology of the eye, a handbook for neurologists and ophthalmologists, vol. i., part ii. Wiesbaden, 1900: J. F. Bergmann. (Reviewed, vol. xxix., 2, p. 256.)

4. Guttmann. The eye diseases of childhood and their treatment. Berlin, 1900: Fischer's medical bookhouse.

5. BERNHEIMER. The region of the nuclei of the ocular nerves. *Graefe-Saemisch Handbuch*, 16th portion, vol. vi. Leipsic, 1900: Engelmann.

6. SCHULTZE, OSCAR. Microscopic anatomy of the lens and zonula. *Ibid.*, 17.

7. GREEFF. Anatomy of the optic nerve and retina. *Ibid.*, 17 and 21.

8. v. HIPPEL. The malformations and congenital anomalies of the eye. *Ibid.*, 18 and 19.

9. WADSINSKY. Methods of examination and forms of eye diseases of the patients in the military hospitals. *Wojenno med. Journ.*, 1899, viii.–ix.

10. COHN. The print of eye journals from a hygienic standpoint. *Ophth. Klinik*, iv., No. 1.

11. KERSCHBAUMER. Sarcoma of the eye. Wiesbaden, 1900: Bergmann. (Reviewed, vol. xxix., 2, p. 254.)

12. HIRSCHBERG. The development of ophthalmology in the 19th century. *Berl. klin. Wochenschr.*, 1900, Nos. 3 and 4.

13. SCHMIDT-RIMPLER. Retrospect of a quarter century of ophthalmology. *Deutsche med. Wochenschr.*, 1900, No. 1.

14. WINDMÜLLER, ERNST. The ophthalmology of Alcoatim (1159). Fourth part. *Inaug. Dissert.*, Berlin, 1899.

15. SCHWARZWEISS, LEO. *Ibid.* Fifth part. *Ibid.*

16. ALLARD, EDUARD. *Ibid.* Sixth part. *Ibid.*

17. KAEMPFER, REINHOLD. *Ibid.* Seventh part. *Ibid.*, 1900.

18. BRONNER, WOLF. The ophthalmology of Rhases. *Inaug. Dissert.*, Berlin, 1900.

19. BRECHT. Annual report of the policlinic. *Charité-Annalen.*, xxiv., p. 368.

20. BROOKLYN EYE AND EAR HOSPITAL. Thirty-first annual report, 1899.

21. PRESBYTERIAN EYE, EAR, AND THROAT CHARITY HOSPITAL OF BALTIMORE. Twenty-second annual report, 1899.

22. EPISCOPAL EYE, EAR, AND THROAT HOSPITAL OF WASHINGTON, D. C. Third annual report, 1899.

23. MANHATTAN EYE AND EAR HOSPITAL. Thirtieth annual report, 1899.

24. NEW YORK OPHTHALMIC AND AURAL INSTITUTE.

Thirtieth annual report, for the year ending Sept. 30, 1899. H. Knapp and others.

25. WELLS, D. W. Sight and hearing of the school children of Wellesley, Mass. *Journ. of Education*, Feb. 15–22, 1900.

26. COLLINS, TREACHER. The Erasmus Wilson lecture on the anatomy and pathology of the eye. *Lancet*, Feb. 17–24, 1900.

In the third edition of HAAB'S (1) atlas of ophthalmoscopy, nine drawings have been added, so that now the number of plates is 80.

GUTTMANN (4) in his book gives a short, plain presentation of the more important eye diseases of childhood. He discusses particularly the connection between eye diseases and general diseases, external conditions and school arrangement. Etiology, prophylaxis, and therapy are treated extensively, rendering the book valuable for the general practitioner.

v. HIPPEL'S (8) work is an exhaustive treatment of the malformations and congenital anomalies of the eyes, taking up first the malformation of various parts of the ball, then those of the entire ball, those of the lids, teratomata and dermoids of the ball, and, finally, anomalies of the lachrymal apparatus and color anomalies. We have now a comprehensive presentation of the subject.

WINDMÜLLER'S (14) dissertation contains the original text of Tractatus IV. of the ophthalmology of Alcoatim, an Arabian physician, whose work appeared in the year 1159. This portion is the pharmacological and therapeutical part.

Tractatus V. is taken up by SCHWARZWEISS (15). This contains the recipes for the innumerable collyria, powders, and salves.

Tractatus VI., taken up by ALLARD (16), contains recipes for dry eye remedies, which even in Alcoatim's day had long been in use.

KAEMPFER'S (17) dissertation naturally follows the last. It is a curious collection of all sorts of recipes for remedies to be used in the various diseases of the eye.

BRONNER (18) reports on the ophthalmology of Rhases, an Arabian physician of the 9th century. There are only seven of his books preserved, which treated of philosophy, astronomy, and chemistry as well as of medicine. The ninth book, devoted to special pathology, contains also an exposition of the diagnosis and therapy of eye diseases. It is this book which forms the subject of this thesis.

During the year 1899, there were treated at the Brooklyn Eye and Ear Hospital (20) 10,224 new eye patients. There were 1535 operations, of which 24 were simple extractions and 10 with iridectomy. BURNETT.

During the year 1899, 6832 new eye cases were treated at the Baltimore Eye, Ear, and Throat Hospital (21). There were 2361 operations on the eye, all told. There were 56 simple extractions and 31 with iridectomy. BURNETT.

During 1899 there were treated at the Eye, Ear, and Throat Hospital of Washington, D. C. (22), 987 new patients; 148 operations were done, of which 24 were extraction of cataract: 18 successes, 5 partial, and 1 failure. BURNETT.

The number of new eye patients treated at the Manhattan Eye and Ear Hospital (23) during 1899 was 13,251. There were 3126 operations, of which 89 were simple extraction and 18 Gräfe. BURNETT.

During the year ending Sept. 30, 1899, there were treated at the New York Ophthalmic and Aural Institute (24) 9434 new eye patients. There were 454 patients admitted to the hospital. There were 590 operations, the minor ones not counted; there were 125 extractions of cataract, 104 simple with one failure, 21 with iridectomy with one failure, 8 congenital (5 zonular), and 72 secondary cataracts were discinded; no failure. BURNETT.

WELLS (25) has examined the eyes of school children of Wellesley, Mass., 685 in number, and found 22 % had normal eyes optically, 63 % H. and H. astig.; 9 % M. and M. astig. The myopia was 7 % in the primary grade, increasing to 12 % in the high and grammar schools; 70 % of H. in the primary and 54 % in the high schools. BURNETT.

COLLINS (26) takes up some of the chapters from the anatomy and pathology of the eye with special reference to embryology and comparative anatomy. The subjects treated are copiously illustrated with the results of original investigations and by the description of partly personally observed cases. The scientific importance of these lectures is such as to demand the study in the original and makes an adequate abstraction extremely difficult. Hence, an attempt will only be made to mention a few of the subjects and conclusions.

Xerosis of conjunctiva and cornea, due to the lack of moisture, and a cessation of the constant friction of the lids over the front of the eye in the act of winking, is an example of the ocular

mucous membrane taking on the character of the skin. In a case examined microscopically the superficial epithelium was not only found thickened, but there were numerous down-growing finger-like processes of epithelium. Dryness of the cornea after instillation of cocaine is due to the retraction of the eyelids from stimulation of the unstriated fibres of Müller. In glaucoma, the following changes are observed in the cornea according to the degree and deviation of the increased intraocular pressure: œdema; vesicles, composed of a loose network of fibres and branching cells between the epithelium and the ant. limiting layer; and the formation of fibrous tissue in this location, with processes breaking through the ant. limiting membrane. This fibrous tissue in depth of anterior corneal epithelium is similarly found in the transverse film of the cornea, and is probably brought about in the same way. Its typical position in the cornea is the result of the pressure of the lids on the cornea. The filamentary processes observed in filamentary keratitis are explained by their being the vertical ridge of cells remaining between two closely adjoining vesicles, which comes to the surface when the vesicles break. As the posterior epithelium of the cornea has been seen to produce in pathological cases a substance akin in structure to the hyaline layer of Descemet's membrane, it is now believed that the latter structure is normally derived from its endothelium. A case of the formation of a hyaline membrane on the anterior surface of the iris under the endothelium is described. The relative size of the cornea to the eyeball in man, compared with other mammals, shows that in man the cornea relatively grows smaller as the size of the eyeball increases, hence the space occupied by the filtration angle lessens relatively to the amount of contents in the globe. The topography of the parts about the angle of the anterior chamber also changes. The similarity in structure of the fibres of the ligamentum pectinatum to the hyaline layer of Descemet's membrane makes it probable that they are the product of the endothelium lining the space of Fontana. In cases of congenital glaucoma, the structure of the ligamentum pectinatum and relation of parts about the iris angle seem to have remained in the fœtal condition and resemble that met in the lower animals. Bands of adhesions across the iris angle were found in some of these cases. As evidence that the ciliary body is the only source of intraocular fluid, the fact is brought forward that the intraocular pressure is not reduced when any of the other vascular structures

of the eye are excluded, as in total absence of iris, embolism of central retinal artery, complete congenital absence of choroid,[1] and after optico-ciliary neurectomy.

After describing the comparative anatomy and embryology of the ciliary body, the examination of a microphthalmic eye is given where the fœtal vascular system persisted and the ciliary processes were absent. In microphthalmic eyes with cystic protrusions, Collins found nothing in the eyes examined pointing to an origin inflammatory in nature. A discussion of the occurrence of elastic fibres in the sclera follows. In a microphthalmic eye with malformations, Collins found hyaline cartilage in the sclera. Cystoid and bulging scars near the sclero-corneal junction after operations are of these kinds. The first, the cystoid cicatrix of v. Graefe, is a gap in the sclero-corneal tissue covered by conjunctiva and lined by a piece of atrophied iris. Collins is convinced that to form a permanent fistula in the sclera the edges of the wound must be lined by cells of epithelial character, otherwise they would unite. This lining may be furnished by the iris, or the ciliary process, or Descemet's membrane with its endothelial cells. In the second, a bulging scar lined by atrophic iris exists when a part of the sclero-corneal wound unites before increase of intra-ocular pressure takes place ; necessarily no filtration is possible. The third, called a staphylomatous scar, occurs when the whole length of iris becomes prolapsed and the sclero-corneal tissue cannot reunite. The whole iris becomes considerably distended and forms a large grayish prominence. A typical case of panophthalmitis in an eye with a cystoid scar is given, and attention is drawn to the possibility of a serous or plastic inflammation befalling an eye with a cystoid scar or subconjunctival entanglement of iris. Two cases are reported. Collins believes that this possibility may explain the occurrence of sympathetic ophthalmia a long time after injury. Sympathetic ophthalmia may occur after operation of abscission, as in the case reported, where portions of the ciliary processes were found entangled in the scar and the site of inflammatory changes. The chance of subconjunctival entanglement of iris or ciliary body forming the path of infection at any time subsequently, likely to incite sympathetic inflammation, occurs to Collins to be perhaps one reason why wounds of the ciliary region are especially liable to be followed by the above complication. The decrease of sympathetic ophthalmia at the

[1] Case reported by A. H. Thompson. *Transactions Ophth. Soc. U. K.*

present day may be due to greater care being taken to avoid entanglement of the iris during operations and to remove any incarceration of uveal tract when present. Prolapse or tags of vitreous may also form the channel for infection. Membranes after cataract extraction, often interfering with sight, are due to a folding of the anterior lens capsule, to which is added any one or more of the following three conditions: (1) Retained lens substance, which becomes opaque and shut off from further absorption by proliferation of the capsule cells. (2) New growth of capsule cells. These cells proliferate and form large vesicular cells, or they may lengthen out into spindle shapes and into delicate fibres which form a dense laminated structure. This tissue is like that found in ant. polar cataracts, and chiefly occupies the folds of the anterior capsule. (3) Adventitious fibrous tissues may form from cellular exudate in iritis on anterior surface or in cyclitis on posterior surface. ARNOLD KNAPP.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

27. v. AMMON. Diagnosis and therapy of ophthalmia neonatorum. *Münch. med. Wochenschr.*, 1900, No. 1.

28. BRECHT. Clinical contributions to the bacteriology of conjunctivitis. *Charité-Annalen*, xxiv., p. 368.

29. HAUENSCHILD. The bacteriology of conjunctivitis with special reference to school epidemics. *Zeitschr. f. Augenheilk.*, iii., p. 200.

30. GROMAKOWSKI. A contribution to the bacteriology of follicular diseases of the conjunctiva. *Arch. f. Augenheilk.*, xli., p. 197.

31. HESS. On congenital ocular cysts and their development. *Ibid.*, p. 1.

32. LANGENDORFF. On the relations of the superior cervical ganglion of the sympathetic to the eye and the blood-vessels of the head. *Klin. Monatsbl. f. Augenheilk.*, xxxviii., p. 129.

33. NICATI. A note on intraocular tension and the mechanism of the blood pressure in the capillaries. *Compt. rend. de l'Académie des Sciences*, Dec. 11, 1899.

34. BÄUMLER. On the question of deep infectious injuries of the eye. *Klin. Monatsbl. f. Augenheilk.*, xxxviii., p. 25.

35. HAUENSCHILD. Investigations on the action of the newer

antiseptics on infected wounds of the cornea. *Münch. med. Wochenschr.*, 1900, p. 146.

36. VOGEL, H. A contribution to experimental investigations on the entrance of soluble substances into the interior of the eye by diffusion after subconjunctival injection. *Graefe's Archiv*, xlix., p. 610.

37. GRÖNHOLM. Experimental investigations on the action of eserine on the exchange of liquids and the circulation in the eye. *Ibid.*, p. 620.

38. SCHMITZ. A test for simulation by using mirror writing. *Wochenschr. f. Ther. u. Hyg. d. Auges*, 1900, p. 162.

39. OHLEMANN. On balneotherapy in eye diseases. *Ibid.*, iii., No. 21.

40. HERTEL. Hydrotherapy in eye diseases. Text-book of clinical hydrotherapy. Jena, 1900: E. Fischer.

41. EALES. Ocular phenomena associated with Cheyne-Stokes respiration. *Lancet*, Feb. 24, 1900.

42. BULL, C. S. The significance of intraocular hemorrhage as to prognosis of life. *Med. Record*, Feb. 3, 1900.

In a hundred cases of ophthalmia of the new-born, V. AMMON (27) found the gonococcus but 56 times. Most of these were cases of late infection, from which it may be concluded that Crede's method cannot particularly diminish the number of cases. The pneumococcus plays an important rôle. The suppuration in such cases ceases suddenly after 3–5 days. According to v. Ammon's investigations neither nitrate of silver solutions nor protargol enter the tissues deeply enough to destroy the micro-organisms. He recommends the use of cold applications and washing out the conjunctival sac with physiological salt solution. After 3–5 days astringents may be used, protargol 10–20 per cent. being of value.

BRECHT (28) reports on a child with severe conjunctivitis, in the secretion of which he found pneumococci and later diphtheria bacilli. The child died of nasal diphtheria. He described also a case of benign conjunctivitis with pseudo-gonococci, a case of gonorrhœal conjunctivitis and rhinitis with gonococci in the nasal pus, and a case of gonorrhœal conjunctivitis which ended in panophthalmitis, necessitating enucleation. Suddenly an acute hemorrhagic nephritis developed which Brecht regarded as being due to the entrance of gonococci into the circulation.

HAUENSCHILD (29) examined 25 cases of acute conjunctivitis and found the pneumococcus 13 times. He gives the well-known symptoms of pneumococcus conjunctivitis. In a school epidemic he found the pneumococcus 8 times in 30 cases.

HESS (31) states that the congenital cysts of the eyeball arise as follows: The invagination and closure of the secondary vesicle take place in the normal manner. At the point of union the wall of the eyeball is not sufficiently resistant, and, yielding under the intraocular pressure, bulges out. The outer layer of the secondary optic vesicle follows this bulging and lines the inner surface of the cyst.

From a series of experiments, mostly on cats, LANGENDORFF (32) has found that after excision of the cervical sympathetic, the myosis, narrowing of the palpebral aperture, and sinking in of the ball may persist for years. After the excision the symptoms of paralysis of the sympathetic, particularly the myosis, are well marked. They gradually grow less marked and may entirely disappear or even pass in the other direction. In all cases this may be produced several days or weeks after the operation by narcotizing the animal or by sensory excitation; perhaps dyspnœa has the same effect.

The investigations of NICATI (33) were in regard to the relations of the intraocular tension to the atmospheric pressure. Under the pressure pump the intraocular tension increases in the rabbit. The latter effect is less marked in the adult than in the young.

BÄUMLER (34) treated three cases of deep infected injuries of the eye with the galvano-cautery, with good results.

HAUENSCHILD (35) has experimented with oxycyanide of mercury, nitrate of silver, carbolic acid, and protargol. The cyanide of mercury is a good antiseptic, but protargol did not come up to expectations.

VOGEL (36) made subconjunctival injections of bichloride of mercury, cyanide of mercury, and other mercurials, and found in the aqueous humor traces of mercury which at the most would have made a 1:100,000 solution. This naturally could have no therapeutic action.

HERTEL (40) discusses fomentations and douches, and the special indications for hydrotherapy in various ocular affections.

In a case exhibiting Cheyne-Stokes respiration reported by EALES (41) the pupils were stationary and somewhat contracted

during the interval. After the onset of the first four or five respirations each pupil began to dilate, and as the respirations increased in volume and rapidity they each became more and more dilated until finally they became widely dilated and remained stationary during the height of the cycle. As the respirations subsided during the decline of the cycle, each pupil gradually contracted more and more, until eventually, just before the respirations ceased, they became contracted somewhat and remained stationary till the onset of the next cycle. ARNOLD KNAPP.

In the young, according to BULL'S (42) observation, hemorrhages into the conjunctiva, and even the retina, are not of grave significance. When, however, it passes into the vitreous it points to general vascular degeneration. In the old, however, retinal hemorrhages are of serious prognostic significance, especially as to apoplexy. BURNETT.

III.—INSTRUMENTS AND REMEDIES.

43. DARIER. Ocular analgesics. *Bull. de la Soc. d'opht. de Paris*, Mar. 5, 1900.

44. PERGENS. Argyrosis of the conjunctiva from the use of protargol. *Klin. Monatsbl. f. Augenheilk.*, xxxviii., p. 256.

45. PICK. Remarks on the action of peronin on the eye. *Ibid.*, p. 45.

46. NEUSCHÜLER. The acetate of cocaine in cauterizations with nitrate of silver. *Suppl. al Policlinico*, 1900, No. 16.

47. SCRINI and ARTAULT. Nirvanin in ophthalmology. The value and the preservation of its preparations. *Arch. d'opht.*, xix., 12, p. 723.

48. AXENFELD. A new Westien binocular hand-loupe for preparing specimens for clinical purposes, with changeable pupillary distance and adjustable head-holder. *Klin. Monatsbl. f. Augenheilk.*, xxxviii., p. 20.

49. LANDOLT. A new stereoscope for re-establishing binocular vision. *Arch. d'opht.*, xix., 12, p. 689.

50. BERGER. Transformation of the simple loupe into a binocular and stereoscopic loupe. *Rev. génér. d'opht.*, 1900, No. 3.

51. PARENT. A new model of spectacles for patients who have been operated on for cataract. *Bull. de la Soc. d'opht. de Paris*, Feb. 6, 1900.

52. BONDI. A portable dark room with illuminating apparatus for ophthalmoscopic purposes. *Wiener med. Presse*, 1900, No. 4.

53. HINGINGA. Eviscero-neurectomy — a new operation. *Jour. Amer. Med. Assoc.*, Feb. 17, 1900.

54. HUBBELL. The Maddox rod or phorometer, which? *Ibid.*

55. MEYERS. Electrolysis in granular disease of the eyelids. *Ophth. Record*, Jan., 1900.

56. MURDOCK. A note on ovoid glass balls. *Amer. Jour. of Ophth.*, March, 1900.

57. ROY. Subconjunctival injections in certain diseases of the eye. *International Clinics*, IV., Ninth Series.

58. STEPHENSON. The practical application of largin in diseases of the eye. *British Med. Jour.*, March 17, 1900.

DARIER (43) employed acoin and dionin in order to produce analgesia in painful diseases of the eye. A little of the drug in powder form is introduced into the conjunctival sac, where at first it causes burning, chemosis, and swelling of the lids, which require the use of a bandage. In three cases of iritis the pain was entirely relieved in this way. In iridochoroiditis the pain was not relieved. In various sorts of corneal disease the tension was reduced and pain relieved. Peronin and heroin are of less use in ophthalmology. BERGER.

PERGENS (44) calls attention to the frequency with which argyrosis follows the use of protargol.

PICK (45) instilled a one to two per cent. solution of peronin. Immediately there was severe burning that reached its acme in four or five minutes. At the same time chemosis appeared, at first with injection and later without, the white swollen conjunctiva being elevated, wall-like, about the cornea. The pupil at first dilated and after fifteen minutes began to contract. An almost complete anæsthesia of the cornea began in the course of a minute and lasted half an hour.

To anæsthetize the cornea before using nitrate of silver NEUSCHÜLER (46) uses five per cent. solution of nitrate of cocaine, as the usual hydrochlorate is precipitated by nitrate of silver and rendered ineffective. KRAHNSTÖVER.

According to the investigations of SCRINI and ARTAULT (47) nirvanin is inferior to cocaine, notwithstanding that it is less

poisonous and does not dilate the pupil, since it irritates the eye, and even in strong solutions does not always produce anæsthesia. It does not possess the antiseptic qualities attributed to it, for carefully prepared aseptic solutions soon contain micro-organisms.

v. MITTELSTÄDT.

In LANDOLT'S (49) stereoscope constructed for the purpose of facilitating binocular vision the stereoscopic figures are photographed upon transparent glass discs. The stereoscope is turned toward the sky, so that the image seen by the weaker eye is illuminated as much as possible, while the image seen by the other eye can be dulled with gray glasses until the visual impressions in the two eyes are of equal intensity and fusion is facilitated.

v. MITTELSTÄDT.

The portable dark room described by BONDI (52) consists in a screen which can be folded up. This is spread about the patient and the examiner, resting on their shoulders. The illuminating apparatus is a head-band with an accumulator placed on the patient's head with incandescent lamps to either side.

HINGINGA'S (53) modification of the operation of evisceration consists in adding an excision of the posterior part of the globe, including a portion of the nerve, through the anterior opening left after the excision of the cornea. A Mules glass globe can be applied if desired. The dangers of sympathetic ophthalmia are thought to be lessened. BURNETT.

In a comparative experience of several hundred cases of all forms of heterophoria HUBBELL (54) finds the Maddox rod test more satisfactory and reliable than the phorometer.

BURNETT.

MEYERS'S (55) more extended experience in the use of electrolysis in granular disease of the eyelids since his first publication in 1891 has been very satisfactory, and he has now abandoned the use of any drug applications. Three or four punctures are made in each hypertrophy, and as many as twenty or thirty punctures may be made at a sitting. A current of one and a half to two milliamperes is sufficient. BURNETT.

MURDOCK (56) uses balls of glass, ovoid in shape, which he places in the socket after enucleation, after the stump has healed, and over this fits the artificial eye. He claims this relieves much the sunken appearance of the orbit. BURNETT.

After an experience of three years with subconjunctival injections ROY (57) believes that in infected processes of the cornea,

such as ulcers, etc., the method is quicker than the treatment usually employed. In chronic iritis it is better than in the acute form. In post-operative infection and panophthalmitis it is the best method we have. BURNETT.

STEPHENSON (58) has experimented with largin, a new substitute for nitrate of silver, and comes to the following conclusions: "The application of largin even in concentrated form is painless, but when prolonged beyond a few weeks may stain the conjunctiva. It acts well in blepharo-conjunctivitis and in some cases of dacryocystitis. It is an efficient substitute for silver nitrate in any of the conjunctival inflammations associated with the Koch-Weeks bacillus, such as acute infectious ophthalmia and acute or subacute trachoma. It acts admirably as a temporary remedy after any of the operations commonly practised for the relief of chronic trachoma. In gonorrhœal ophthalmia, on the contrary, it is in my experience distinctly inferior both to protargol and to silver nitrate. In diplobacillary conjunctivitis, too, it does not succeed so well as zinc sulphate. In short, largin seems likely to gain a permanent place among the somewhat restricted number of remedies employed in every-day eye work."

ARNOLD KNAPP.

(*To be continued.*)

XIII. — **Der Pemphigus u. die essentielle Schrumpfung der Bindehaut** (Pemphigus and the Essential Shrinkage of the Conjunctiva; a clinico-critical study). By Dr. E. FRANKE, Hamburg. Wiesbaden: J. F. Bergmann, 1900.

Nicely gotten-up 8vo of 111 pages, with a complete bibliography (119 numbers). In the historical introduction it is stated that WHITE COOPER [1] was the first to describe this rather rare and pernicious disease of the conjunctiva in a case of pemphigus vulgaris of the skin. WECKER [2] came out with another case ten years later, drawing also attention to a picture of disease which STELLWAG had published before that time in his *Lehrbuch der pract. Augenheilkunde* under the designation of syndesmitis degenerativa, supposed by Wecker to be the same affection. Again, ten years later PLFÜGER [3] collected the observations published up to that time, adding a few new cases. In the same year appeared an elaborate paper by ALFRED GRÄFE,[4] with the heading Essential Shrinkage of the Conjunctiva, which name he chose because his own cases, showing the same picture of disease, were not accompanied by pemphigus elsewhere. STEFFAN [5] protested against the separation of the same picture of disease complicated with affections of skin or mucous membranes from those where the conjunctiva alone was affected. Most German ophthalmologists up to the present time are on the side of Steffan, for instance SCHMIDT-RIMPLER [6] in his excellent treatise, whereas many dermatologists do not agree with them at all. UNNA,[7] in particular, emphasizes sharply that pemphigus of the skin may naturally give rise to similar affections of mucous membranes, but that a series of diseases described under the name of pemphigus conjunctivæ

[1] *Ophth. Hosp. Rep.*, 1858.
[2] *Klin. Mon. f. Aug.*, 1868.
[3] *Ibid.*, 1878, p. 1.
[4] *Arch. für Ophthal.*, 1878.
[5] *Klin. Mon. f. A.*, 1884.
[6] *Die Erkrankungen des Sehorgans im Zusammenhang mit Allgemeinen Krankungen*, Wien, 1898.
[7] *Histopathologie der Hautkrankheiten*, Berlin, 1894.

had nothing in common with pemphigus. In the earlier literature of the nineteenth century the disease has been described by many authors under the name of xerophthalmus, and was mostly considered as an extreme degree of conjunctival atrophy caused by the trachomatous process. Of such cases the reviewer has seen a certain number with ulcers without formation of vesicles, but also a few, all binocular, where there was a vesicular disease in the nose, pharynx, and mouth. After reporting his own (5) cases, the author gives extracts of all published cases (102). Then he reviews the material on hand. He finds formation of vesicles only in 14 cases; UHTHOFF thinks it to be present in 25 per cent. The seat of the vesicles is in the superficial layers of the epithelial stratum, *i. e.*, intra-epithelial. In a certain number it may also be deeper, so as to run its course without or with formation of scars. The characteristic sign of the disease is shrinkage of the conjunctiva, which is followed by partial or total symblepharon, as well as by both entropium and ectropium, occupying different places of the same eye; finally there will be more or less extensive ulceration, destruction, and cicatrization.

The most frequent complication is skin disease, 74 per cent., of the same nature.

The pathology is still insufficiently cleared up. SATTLER (Leipzig) found the whole sub-epithelial connective tissue infiltrated with a molecular mass, which accounted for the peculiar white-waxy aspect of the surface, but he failed to define the nature of this infiltration. GELPKE says that the membranes of the blisters consist of coagulated fibrine with enclosed leucocytes. BÄUMLER found in transverse sections of a hardened specimen, the anterior parts of the eye and lids (which had been removed together) without notable alterations.

DEUTSCHMANN found in the conjunctival tissue xerosis bacilli and small cocci resembling those contained in pemphigus vesicles, conditions to which the author denies any etiological significance, as LÖFFLER and other exact observers have failed to detect any bacteria in the liquid contents of the first vesicles.

The nosology of the disease is not clearly known. Even dermatologists, for instance NEISSER, pretend that the term pemphigus is still ambiguous, referring to different diseases, yet all authors agree that its essential feature is the presence of superficial vesicles without any or with only extremely insignificant inflammatory products in the deeper strata. In 26 out of the 101 well-constituted

cases the process was confined to the conjunctiva, in the remaining 76 the skin of the lids also was affected. In concluding this chapter the author says: "As long as dermatologists are not united as to the nature of pemphigus, we should replace this word by a clinical designation, adopting in the first place the essential shrinkage of the conjunctiva of Alfr. Gräfe, or, perhaps better still, we might call it *shrinkage of the conjunctiva with formation of vesicles.*"

The prognosis is unfavorable (in our opinion — hopeless) *quoad visum*, and the treatment powerless. Collyria and operative procedures thus far have failed. The reviewer would say to his younger colleagues: There is work to be done in this desperate disease, and laurels to be gathered. We should begin with learning how to diagnosticate the affection in its early, possibly curable stage. We have to appeal to the general practitioner and to the dermatologist to give us opportunity to see cutaneous and mucous-membrane cases of pemphigus, and examine them with regard to ocular complications. To such as are willing to do this work, but not to such only, the elaborate monograph of Dr. E. Franke will be of great service. H. K.

XIV.—**Die operative Beseitigung der durchsichtigen Linse** (The Operative Removal of the Transparent Lens). By Dr. E. PFLÜGER, Professor of Ophthalmology, University of Berne. Wiesbaden: Bergmann, 1900.

This monograph of 202 large-8vo pages, with an elaborate table of 101 cases, is the most extensive publication on the above subject, chiefly applied to the operative cure of excessive myopia. It is a German edition of a paper which appeared first in the *Comptes Rendus* of the French Ophthalmological Society. The bulk of the work consists in a description of each case operated on by the author, so that the reader may acquire a complete clinical experience (so far as reading can bestow) of this modern expansion of the cataract operation. Comprehensive presentation and discussion of all the questions on this subject make the long brochure most instructive. H. K.

XV.—**Schädel und Auge** (Skull and Eye). A Study on the Relations between Anomalies of the Conformation of the Skull and the Eye. By Dr. FRITZ DANZIGER. With 7 figures on 3 plates. Wiesbaden: J. F. Bergmann, 1900. Mk. 2.80.

Fifty-six large-octavo pages, well printed, with a minimum of investigation and a maximum of speculation, introduced by four lines from Homer's *Iliad*, in Greek, not translated.

"Refraction, bulbus, and orbit are in a certain relation. The attempt to prove this proposition by measurements of the dimensions of the orbit has failed, but I have obtained by measurements of the skull the sure, positive result that refraction is in a definite relation to the conformation of the skull." The author considers the cause of the anomalies of refraction by a multitude of specious arguments. The hyperopia and the strabismus of the new-born depend upon a malformation of the skull brought about by compression of the skull during the passage of the head through the bony pelvis, which reduces the longitudinal diameter either alone or more strongly than the other diameters. Hence the hyperopia of babies, the most frequent cause of convergent strabismus, which disappears when the deformity gradually gives way to a more physiological shape of the head. In a similar way the anomalies of refraction are examined and accounted for, together with the mechanical causation of keratoconus, posterior staphyloma, and detachment of the retina and the vitreous, nystagmus, accommodation (spasm, paralysis), excavation and atrophy of the optic nerve. The seven figures are all outline drawings which in reduced dimensions might just as well have been put into the text as placed ostentatiously on three large plates. "All rights [as to reprints and translations] reserved." (!) H. K.

XVI.—**Physiologic Optics,** Dioptrics of the Eye, Functions of the Retina, Ocular Movements, and Binocular Vision. By Dr. M. TSCHERNING, Adjunct Director of the Ophthalmological Laboratory at the Sorbonne of Paris. Authorized Translation from the Original French Edition, Specially Revised and Enlarged by the Author. By CARL WEILAND, M.D., Philadelphia. With 212 illustrations. The Keystone: Philadelphia, 1900.

This is a very good school-book of 337 pages, treating of the practical part of the optics of the eye. Tscherning is a well-known scientist in this line, and C. Weiland had no difficulty in clothing Tscherning's work in good English; he would have had none if the book were loaded with mathematics. The author has, however, refrained from dealing much in mathematics: the simplest algebra and arithmetic, with a few geometrical constructions, are all he requires to make himself understood. The Keystone has done many an oculist a great service to bring out so useful a book, and the translator deserves credit for his correct rendition and easy style, evidence that he travelled over familiar ground. H. K.

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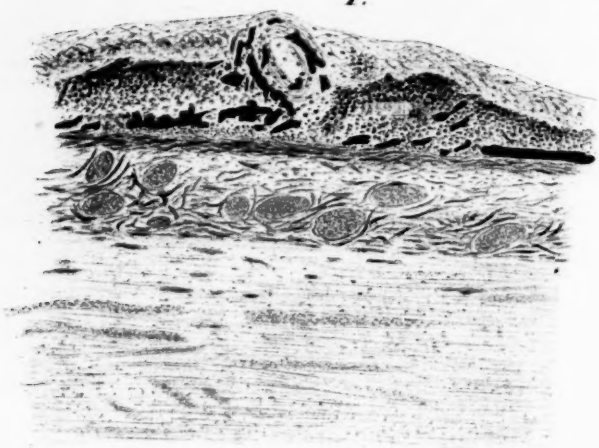

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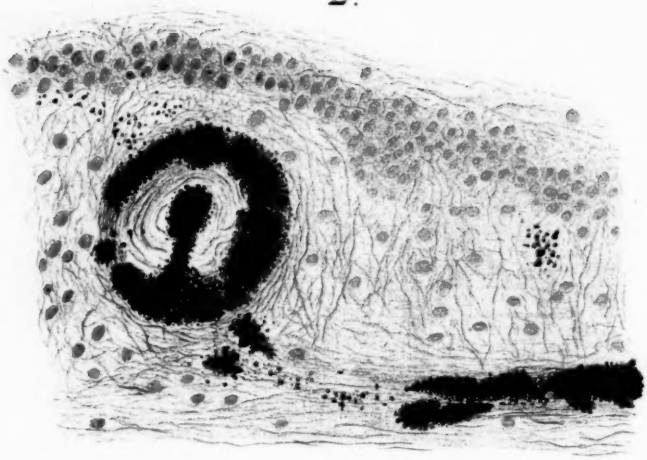

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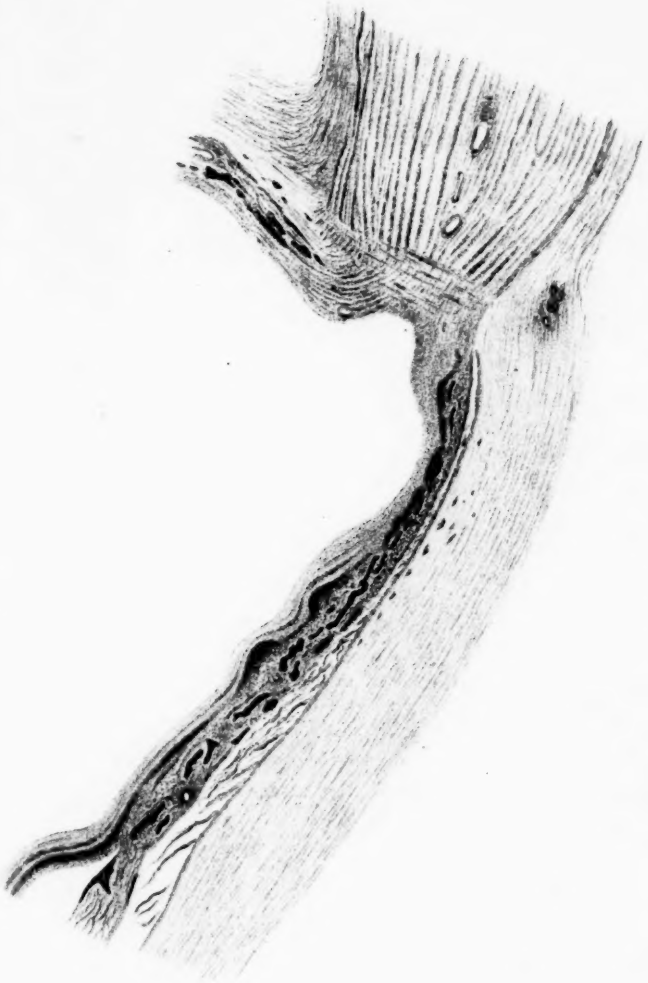

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